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PARAVAGINAL HAEMATOMATA

BASIL BLOCH, M.B., CH.B., M.MED. (O.&G.)

Registrar, Division of Obstetrics and Gynaecology, University of Cape Town and Cape Provincial Administration

Together with many other gynaecological conditions which are often classified as 'minor', paravaginal haematomata have, in the past decade, fallen into relative obscurity. The main reason for this state of affairs is that the supervention of sepsis, previously a major complication, has, with the advent of antibiotics, become relatively easily surmountable and is no longer dreaded. In spite of this the treatment of haematomata is by no means standardized or clearly established and with this problem in view 14 case histories have been reviewed, together with the literature, in an attempt to assess what form of treatment provides uniformly good results.

Haemorrhage into the paravaginal soft tissue with subsequent haematoma formation is not uncommon and it is sometimes a serious complication in obstetrical practice. It is not generally appreciated that this condition is an important contributor to puerperal morbidity and thus to delayed convalescence. This, together with the fact that the condition is not obvious unless specifically looked for, results in the diagnosis being missed in a large number of cases. Rueff, in 1554, described the first case recorded in the literature, but not until 1830 was a detailed study of the condition undertaken by Beneux[1] and his description was remarkably accurate, even by present day standards. A number of papers on the subject have been published since, the most comprehensive being that by Williams[2] in 1915.

Classification

The classification may be made in general terms, or based on anatomical considerations. An anatomical classification is more satisfactory than a general classification.

General. 1. Associated with pregnancy and labour, (*a*) before delivery, and (*b*) puerperal.

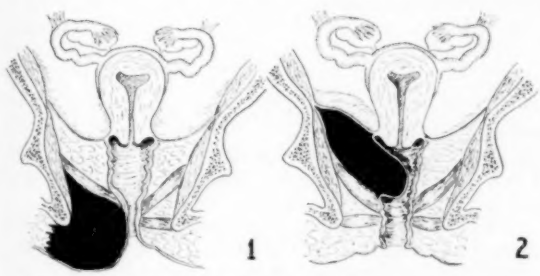
2. Occurring in the non-pregnant female, usually as a result of trauma.

Anatomical. 1. Paravaginal (Fig. 1). (*a*) infralevator, (i) perineal, and (ii) labial; (*b*) supralevator (Fig. 2).

2. Intraligamentous—usually associated with rupture of the uterus.

3. Retroperitoneal.

4. Any combination of the abovementioned groups.



Figs. 1 and 2. Infralevator and supralevator haematomata.

Incidence

The puerperal variety is the commonest, the incidence being variously estimated as 1 in 1,500—2,000,[2] 1 in 4,000[3] and 1 in 135.[4] It is impossible to assess the true incidence because many small tumours pass unnoticed. In all probability it is in the region of 1 in 1,500—2,000.

Anatomy

The paravaginal space is that division of the pelvic connective tissue which extends from the vagina to the obturator fascia. It is continuous with the paravesical, pararectal and parametrial spaces. It follows that any collection of fluid may spread from one compartment to the other. A venous plexus surrounds the vagina, being most prominent along its lateral aspects, because of the condensation of the vesico-vaginal and recto-vaginal fascia. One or more vaginal veins issue from its upper end on both sides and terminate in the hypogastric veins. The vaginal venous plexus intercommunicates with the inferior and middle haemorrhoidal veins and also with the inferior vesical plexus. This entire communicating venous system is tremendously engorged in pregnancy and it is the rupture of one or more of these veins in the vaginal plexus which leads to the formation of a haematoma. The levator ani muscles divide the paravaginal space into supralevator and infralevator fossae. The haemotoma is typically confined to the upper or lower compartment, although a very large haemorrhage can disrupt the muscular barrier.

While we are primarily concerned with the paravaginal type of haematoma, it is as well to remember that any fluid effusion into connective tissue tends to track along the natural planes of cleavage of fascia. When this process occurs in the supralevator fossa, the blood tumour may spread into or below the broad ligament, the perivesical space, or beneath the vesico-uterine fold of peritoneum. These haematomata tend to dissect under the peritoneum and may extend anteriorly under the inguinal ligament or posteriorly to the perirenal or subdiaphragmatic spaces.

The time of appearance of these haematomata varies greatly. They usually occur immediately after delivery or trauma but may require not only hours, but days and even weeks for their full formation. The longest interval recorded between delivery and diagnosis is 14 days.[5]

Aetiology

The primary factor in the causation of this condition is rupture of a blood vessel, usually a vein, and although often no specific aetiological factors can be identified, a number of possible conditions and circumstances have been postulated which might have a bearing on the formation of these tumours:

Excessive trauma during childbirth. The majority of these lesions occur in primigravidae, 59%[6] in one series and 76%[7] in another. This has been cited as a factor in favour of trauma being a common aetiological factor. However, since these haematomata occur in short and uncomplicated labours this cannot be the only explanation of their causation.

Improper haemostasis. In order to prevent the occurrence of a haematoma it is of importance, when suturing an episiotomy or perineal tear, that bleeding is arrested and that adequate attention be paid to the proper apposition of the skin edges.

Pressure necrosis of the affected vessels is thought to be the responsible factor in delayed haematoma formation. The fact that these lesions occur with short or average labours and with average or small-sized babies, and that they may be associated with breech presentations where the vertex passes through the pelvis fairly rapidly, indicates that pressure is not always the operative factor.

Pre-eclampsia is said to predispose to paravaginal haematomata because of vascular changes. The lesions are thought to be local manifestations of the pre-eclampsia, analagous to the haemorrhages which occur in the uterine wall in a concealed accidental haemorrhage.[8]

Blood dyscrasias. Interference with the clotting mechanism is said to be the operative factor increasing the incidence of these haematomata. An example given by Lyons[9] is afibrinogenaemia.

Varicosities of the vulval area are associated with an increased incidence of paravaginal haematomata.

Vigorous uterine massage in the treatment of post-partum haemorrhage is incriminated as an aetiological factor by Samuelson.[7]

Prolonged labour, particularly of the second stage, and large babies have been cited as factors causing haematomata.

Trauma, other than during childbirth, in both the pregnant and non-pregnant state is a common aetiological factor.

Diagnosis

The diagnosis is not as a rule difficult provided that the vulval and perineal regions are inspected and a rectal examination performed where indicated. The common reason for missing many of these lesions is that pain in these areas is expected after delivery and therapy is prescribed before ascertaining the cause of the pain. The alert will, however, observe that the pain is often severe, even excruciating. Pain is a prominent feature in larger tumours and is often likened to something tearing. It is not relieved by opiates. The sudden appearance of a tense elastic mass, blue in colour, tender, and encroaching on the vagina in the infralevator variety, is not often missed (Fig. 3). Rectal examination will show a similar mass at a higher level should the supralevator region be affected. A persistent low-grade fever is often present. *Shock* and *anaemia* are features found in large haematomata, particularly in those which have extended retro-peritoneally. Anorectal tenesmus

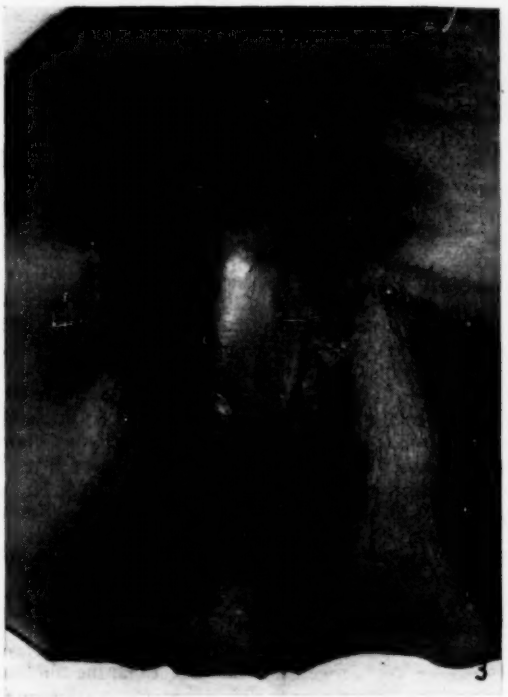


Fig. 3. Tense mass in infralevator haematoma.

results from extension into the ischiorectal fossa. Urinary retention may be due to extension of the mass into the paravesical space. Reflex ileus, pain in the thighs and swelling of the lower limbs due to compression of the venous drainage to the legs, are rarely encountered.

TREATMENT

The earliest recorded treatment for this condition was the conservative or expectant approach. The only indication for incision and drainage was superimposed infection. The next step in the evolution of therapy was an attempt to avoid sepsis by incision and drainage as soon as thrombosis was complete, i.e. after 48 hours. At the present time there is no conformity of opinion regarding the type of treatment which consistently yields good results. The forms of treatment which have been utilized are briefly outlined as follows:

A. *Preventive*

1. *Before delivery.* (*a*) Adequate antenatal care with particular attention to the correction of anaemia, and (*b*) early treatment of pre-eclampsia and recognition of the blood dyscrasias.

2. *At delivery.* (*a*) Avoidance of excessive trauma to the maternal soft tissues, and (*b*) adequate haemostasis and repair of tears and episiotomies.

3. *Post-partum.* Becoming 'haematoma conscious'. This includes early diagnosis and prevention of spread. Where a suspicion of this condition arises, a thorough perineal and rectal examination must be performed.

Preventive treatment applies only to the haematomata which occur during pregnancy.

B. *Active*

1. *During labour.* If a haematoma forms and increases rapidly in size, immediate evacuation is the treatment of choice. Haemostasia must be secured rapidly by the most suitable technique.

2. *After delivery or in the non-pregnant state.* (*a*) Expectant. The measures recommended include: (i) Antibiotic therapy, (ii) ice packs to the affected area, (iii) adequate antisepsis, (iv) compression with binders and T. bandages, (v) sedation with morphia or pethidine, and (vi) elevation of the foot of the bed.

Mengert[10] considers that this form of treatment gives the best results; incision is reserved for those cases where infection becomes superimposed.

(*b*) Surgical—Important points to observe are: (i) Adequate drainage through an incision placed in the vaginal mucosa, (ii) evacuation of blood clots, (iii) adequate haemostasis by ligation of bleeding points, mattress sutures or packing of the cavity, (iv) counter pressure can be made by packing the vaginal canal or even the rectum, (v) replacement of blood loss where indicated, and (vi) antibiotic therapy.

Other therapeutic suggestions include:

1. *Aspiration* through a wide-bore needle. The disadvantages of this type of treatment are that blood clots cannot be evacuated and that there is the danger of introducing infection.

2. *Haemostatic gauze* sutured into the wound. A foreign body is introduced into the wound and may precipitate an infection.

3. *Enzymatic debridement* of the wound using streptokinase and streptodornase.[11]

It is well to remember that exploration of the abdomen and even hysterectomy may become necessary where the haematoma extends above the cardinal ligaments or where spread of the haematoma cannot be checked.

Assessment of the Types of Treatment

In an attempt to assess which types of treatment advocated yield the best results, 14 cases which occurred during the years 1954-56 (inclusive) were analysed. These were all cases admitted as emergencies to the gynaecological wards of Groote Schuur Hospital, Cape Town.

A. Nine cases were associated with pregnancy; of these 5 occurred after birth. The histories indicated normal vertex deliveries. Large perineal lacerations were present. In 4 cases attempts at suture had been made before admission to hospital. The haematomata were present in 4 pregnant patients. The period of gestation in these varied from 12-28 weeks. Two of these had arisen spontaneously, the other 2 were due to direct trauma.

B. Five cases were not associated with pregnancy; 4 were due to direct trauma and one had arisen spontaneously.

In Table I a summary is given of the morbidity and duration of stay in hospital.

TABLE I. MORBIDITY AND DURATION OF STAY IN HOSPITAL

Type of Treatment	*No. of cases*	*Hospitalization (days)*	*Morbidity*
Incision and drainage ..	9	6	1
Conservative	5	11	3

Case Report

A very interesting case which prompted this enquiry was that of a 45-year-old Coloured female who was admitted to hospital with a very large left-sided infralevator haematoma. Conservative treatment was instituted because she was considered a poor anaesthetic risk. After a period of 9 days the haematoma had not decreased in size. Incision under local anaesthetic was undertaken, clots were evacuated and an acriflavine-emulsion plug was inserted into the cavity. The plug was removed on the following day. Five days later the patient was discharged since the wound required no further dressing.

The results in the small series of cases quoted above would appear to support the statement made by Samuelson[7] that 'primary operation and drainage is followed by less infection, shortened hospitalization and more rapid healing than conservative treatment. Early ambulation is not contra-indicated'. The statement that early ambulation is not contra-indicated is important because in Samuelson's series where conservative treatment was employed 2 cases developed thrombo-embolic and respiratory complications.

Prognosis

The maternal mortality in the paravaginal variety is stated to vary between 0-9%-21%[6] and in the retroperitoneal variety 56%-79%.[9] These figures apply to the pre-antibiotics and pre-blood transfusion era and the figure now approximates 0%.[12]

The morbidity is estimated at 37% and the average duration of stay in hospital at 7-8 days.

SUMMARY

(*a*) The types of genital haematomata are discussed and their aetiology, symptomatology, diagnosis and treatment reviewed.

(*b*) The importance of early diagnosis is stressed. Where unexplained severe ano-genital pain, ecchymosis or urinary retention occur and rectal examination is performed, the diagnosis will never be missed.

(*c*) Primary incision and drainage appears to be the treatment of choice from the point of view of period spent in hospital, morbidity and early ambulation.

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Suid-Afrikaanse Tydskrif vir Geneeskunde : South African Medical Journal

VAN DIE REDAKSIE : EDITORIAL

TORAKOSKOPIESE INGREPE OP DIE OUTONOME SENUWEESTELSEL—DIE SOGENAAMDE KUX-OPERASIE

Daar is verskeie redes waarom die gemiddelde Suid-Afrikaanse geneesheer teenoor die prosedure, wat as die Kux-operasie bekend is, ietwat skepties staan. Eerstens is die operasie op die gewone sensasionele manier, wat aan die moderne joernalistiek eie is, deur 'n bekende weekblad aan die publiek voorgestel as 'n wonderbehandeling vir verskeie toestande. Tweedens het Kux self by verskillende geleenthede die prosedure vir so 'n verskeidenheid van toestande (insluitende maag- en duodenumulsera, kardiospasme, angina, hipertensie, long-tuberkulose, asma, icterus catarrhalis, diabetes en selfs leukemie) voorgestel, dat wetenskaplik-georiënteerde geneeshere dit moeilik vind om selfs die helfte van die indikasies te aanvaar. Derdens bots Kux se bevindings en teorieë dikwels met die algemeen aanvaarde menings oor die fisiologie van die outonome senuweestelsel. Al hierdie faktore maak dit vir die geneesheer, wat homself afvra of daar onder al hierdie rook tog 'n vuurtjie brand, moeilik om die werklike waarde van die prosedure (indien enige) te bepaal.

Dr. E. Kux is 'n torakale chirurg wat in die chirurgiese universiteitskliniek te Innsbrück, Oostenryk, werk. By die operasie wat na hom heet, word simpatiese of parasimpatiese senuwees in die borskas deur die torakoskoop deurgebrand. Dit word vir verskillende toestande, waarvan die bekendstes peptiese ulserasie en asma is, toegepas.

Wat peptiese ulserasie betref, pas Kux deesdae die operasie hoofsaaklik vir duodenale ulserasie toe. Maagulserasie word omrede van die moontlikheid van maagkanker gewoonlik nie deur sy operasie behandel nie. By die operasie vir duodenale ulserasie word deur die torakoskoop (gewoonlik onder plaaslike narkose) aan die regterkant die vagus, die simpatiese string (op verskeie plekke tussen omtrent ganglia 4 tot 9) en die nervus splanchnicus major met die galvanokouter deurklief. In 'n minderheid van gevalle is dit ook nodig om aan die linkerkant die simpatikus en splanchnicus, en af en toe ook die vagus, te deurklief. Kux maak daarop aanspraak dat selfs met die eensydige ‚vago-simpatikotomie' die oorgrote meerderheid van die pasiënte dadelik simptoomvry word en dat omtrent 80% (uit 'n reeks van 56 wat oor 'n tydperk van 3 jaar opgevolg is) ook roentgenologies genees is. In sy verklaring vir die sukses van hierdie prosedure beklemtoon hy in verskillende artikels[1-4] soms die een en soms die ander van die volgende faktore:

1. Eensydige vagotomie veroorsaak verminderde afskeiding van maagsuur (in direkte teenstelling met die bevinding van Dragstedt,[5] wat die uitwerking van eensydige vagotomie op suurafskeiding as minimaal beskou), verminderde motiliteit van die maag, en ongelukkig ook pilorospasme.

2. Simpatikotomie en splanchnikotomie onderbreek die pynvesels van die distale maag en duodenum, hef die pilorospasme op wat die vagotomie veroorsaak het, en lei tot toename in die bloedsomloop as gevolg van vasodilatasie.

3. 'n Perifere orgaan, wat van sentrale inmenging vrygestel word, kan as outonome eenheid beter funksioneer.[2]

In hierdie kort uiteensetting kan die verskillende betwisbare punte in verband met die uitwerking van die operasie nie bespreek word nie. Vir ons, altans, is die fisiologiese grondslag nog glad nie helder nie. Die moontlikheid bestaan dat die uitskakeling van pyn deur onderbreking van die bose kringloop van spanning—verhoogde suur-pepsienafskeiding—ulcus—pyn—spanning, die belangrikste faktor is. Kux het self hierdie moontlikheid ondersoek deurdat hy vir sy eerste 400 gevalle net simpatikotomie en splanchnikotomie gedoen het. Omdat die resultate veel swakker was as met die ‚vago-simpatikotomie' wat hy later toegepas het, glo hy dat vagotomie ook 'n belangrike rol speel, en beveel hy gevolglik aan dat sowel die simpatikus as die vagus deurklief word.

Deurkliewing van pynvesels kan meebring dat die viscerale pyn van ander organe in die regter-bobuik, soos die galblaas, ook uitgeskakel word, wat moontlik daartoe kan lei dat ontstekingstoestande van hierdie organe relatief asimptomaties en derhalwe gevaarlik kan wees. Hoewel hierdie gevaar bestaan, bly dit uit die ervaring van ander chirurge met gevalle van simpatektomie vir hipertensie dat dit van min praktiese belang is.

Kux het by 800 operasies vir peptiese ulserasie geen sterfgevalle en min komplikasies gehad.[2] Die feit dat daar in ons land gedurende die jaar sedert die eerste operasie hier uitgevoer is, minstens twee sterfgevalle en verskeie komplikasies voorgekom het, behoort vir diegene wat meen dat die prosedure deur enigeen sonder gevaar uitgevoer kan word, 'n waarskuwing te wees.

Om ten opsigte van peptiese ulserasie op te som: Ten gunste van die operasie is die goeie resultate waarop Kux aanspraak maak, die klein ingreep met kort hospitalisasie, en die geringe gevaar daaraan verbonde. Dit is 'n behandelingsmetode wat tussen interne behandeling en die betreklike groot operasie van gedeeltelike gastrektomie (met onherroeplike verlies van 'n groot deel van 'n belangrike orgaan staan. Teen die operasie moet die onsekere fisiologiese grondslag waarop dit berus, gereken word.

Hoewel niemand daarvan hou om 'n operasie wat op onsekere fisiologiese grondslag berus, toe te pas nie, wil ons aan die hand doen dat die Kux-operasie onder die volgende omstandighede in die behandeling van *duodenale* ulserasie op die proef gestel mag word (maagulsera behoort in die meeste gevalle nie met hierdie metode behandel te word nie): Die diagnose moet roentgenologies bewys wees en ander moontlike oorsake van simptome, soos galblaassiekte, moet uitgeskakel word. Die geskikte geval sou dié wees wat op interne behandeling nie genees nie, of na genesing herhaal, maar tegelykertyd nie kliniese of roentgenologiese bewys van diep penetrasie, uitgebreide fibrose, pilorusstenose

of kwaai bloeding toon nie. Laasgenoemde gevalle behoort deur oop operasie, soos gedeeltelike gastrektomie, behandel te word. Wat die uitvoer van die operasie betref, behoort dit alleen gedoen te word deur iemand wat ervaring van torakoskopie het of wat die moeite gedoen het om die tegniek aan te leer. Ook behoort dit in 'n hospitaal gedoen te word waar fasiliteite vir die diagnose en behandeling van moontlike komplikasies bestaan, dit wil sê, fasiliteite vir roentgenondersoeke, bloedoorgieting, intratracheale narkose en torakotomie.

Dit sou vir die beroep en die publiek in ons land van groot waarde wees as 'n wetenskaplike ondersoek, nie alleen na die resultate nie, maar na die fisiologiese uitwerking van die operasie, deur bevoegde persone, veral in 'n geneeskundige fakulteit, uitgevoer kon word.

In die behandeling van *asma* maak Kux[6] ook op redelike goeie resultate aanspraak. Weer is die fisiologiese grondslag waarop sy operasie berus nie duidelik nie. Asma is berug vir die moeilike beoordeling van die resultate van enige soort behandeling, veral omdat sielkundige faktore die toestand so sterk beïnvloed. Die sukses waarop Kux aanspraak maak, is behaal in gevalle wat deur interne behandeling nie verder verbeter kon word nie. Ander chirurge, wat by oop operasie simpatektomie of vagotomie vir asma gedoen het, het oor die algemeen teleurstellende resultate gehad, maar tog af en toe sukses. Daar is dus moontlik, in uitgesoekte gevalle wat deur 'n internis gediagnoseer en sonder sukses behandel is, 'n plek vir die gebruik van die Kux-operasie as proefneming. Die finale beoordeling van die waarde van die operasie vir asma sal eers na jare gemaak kan word.

Die Kux-prosedure word vir verskeie ander toestande gebruik wat nie hier bespreek kan word nie. Ten slotte moet net daarop gewys word dat dit geweldig vatbaar is vir uitbuiting, te meer omdat die publiek op die oomblik dikwels om die operasie vra en wel vir simptome wat dit nie regverdig nie. Die geneesheer, wat die operasie links en regs vir enige vae buikpyn of amborstigheid toepas, handel nie reg teenoor sy pasiënt of teenoor sy beroep nie.

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THORACOSCOPIC INTERRUPTION OF THE AUTONOMIC NERVOUS SYSTEM—THE SO-CALLED KUX OPERATION

There are several reasons why the average South African doctor is somewhat sceptical about the procedure known as the Kux operation. Firstly, the operation has been introduced to the public by a well-known weekly journal in the usual sensational way as a wonder cure for several conditions. Secondly, Kux himself advised this operation for such a wide variety of conditions—ulcers of the stomach and duodenum, cardiospasm, angina, hypertension, tuberculosis of the lungs, asthma, icterus catarrhalis, diabetes and even leukaemia—that doctors with a scientific orientation find it difficult to accept even one half of these indications. Thirdly, Kux's findings and theories are at variance with the accepted conceptions of the physiology of the autonomic nervous system. All these factors make it difficult for the doctor who wonders whether there is in fact some validity in this procedure to assess its value.

Dr. E. Kux is a thoracic surgeon attached to the surgical clinic of the Innsbrück University, Austria. In the operation which is named after him, sympathetic and parasympathetic nerves in the thorax are severed through a thoracoscope. This procedure is carried out for several conditions, of which peptic ulceration and asthma are the best known.

In peptic ulceration Kux reserves the operation for duodenal ulcers. Ulceration of the stomach is not treated by this method because of the possibility of carcinoma. In the treatment of duodenal ulcer the vagus, the sympathetic chain (in several places between, approximately, ganglia 4 and 9), and the major splanchnic nerve, are severed under local anaesthesia through the thoracoscope by a galvano-coulter. In a minority of cases it is considered necessary to repeat the performance on the left side. Kux claims that most patients immediately lose their symptoms—even after unilateral vago-sympathectomy; and that approximately 80% of a series of 56 cases followed up over a period of 3 years remained free from signs on X-ray examination.

In his explanation for the success of this procedure Kux in a number of articles[1-4] stresses one or more of the following factors:

1. Unilateral vagotomy causes decreased secretion of acid by the stomach (in direct contrast to the findings of Dragstedt,[5] who considers that the effect of unilateral vagotomy on acid secretion is minimal), decreased motility of the stomach, and unfortunately also pylorospasm.

2. Sympathectomy and splanchnicotomy interrupt the pain fibres of the distal part of the stomach and duodenum, terminate the pylorospasm caused by the vagotomy, and cause an increase in the circulation of the blood as a result of vasodilatation.

3. A peripheral organ, when released from central control, can function better as an autonomous unit.

In this brief review it is not possible to discuss the various controversial points regarding the effects of the operation. To us the physiological basis of the operation is not at all clear. The possibility does exist that the exclusion of pain by interruption of the vicious circle, tension—increased secretion of acid and pepsin—ulcer—pain—tension, is the most important factor. Kux himself investigated this possibility—in his first 400 cases he performed only a sympathectomy and splanchnicotomy. Because his results in these cases were not as good as in the cases for which he performed a vago-sympathectomy he believes that vagotomy has a definite place in the treatment, and consequently he advises severance of both the sympathetic nerves and the vagus.

Severing of pain fibres may result in the exclusion of visceral pain from other organs in the upper abdomen, e.g. the gall-bladder, and this may precipitate a dangerous situation in infection of these organs. Although this danger does exist it does not appear to be of too great

significance in the light of the experience of other surgeons after sympathectomy for hypertension.

In 800 operations for peptic ulcer Kux had no deaths and only a few complications.[2] The fact that at least 2 deaths and several complications occurred in this country during the year since this operation was first performed here, should serve as a warning to those who think that the operation is without danger and that anybody can attempt it.

The position regarding the treatment of peptic ulcer can be summarized as follows: In favour of the operation are the good results claimed by Kux, the relatively minor nature of the procedure, and the short hospitalization and the small danger attached to it. It is an intermediate procedure between medical treatment and partial gastrectomy with irreversible loss of a large part of an important organ. Against the operation must be mentioned its uncertain physiological basis.

Although nobody is in favour of carrying out an operation based on an uncertain physiological thesis, we feel that the Kux operation might be tried out as a treatment for duodenal ulcer under the following conditions. (Ulcers of the stomach should not be treated by this method.) The diagnosis must be proved on X-ray examination and other possible causes of symptoms, such as gall-bladder disease, must be excluded. A case which cannot be cured with medical treatment, or which recurs after cure, and which at the same time shows no clinical or X-ray evidence of deep penetration, extensive fibrosis, pyloric stenosis or severe bleeding, would appear to be a suitable case for a trial application of the Kux operation. Cases showing signs of the complications mentioned should be treated by open operation, e.g. partial gastrectomy. The Kux procedure in these trial cases should be carried out only by somebody well versed in thoracoscopy or who has taken pains to learn the technique. Moreover, the operation should be carried out in a hospital where facilities exist for the diagnosis and treatment of possible complications, viz. for X-ray investigations, blood transfusion, intratracheal anaesthesia and thoracotomy.

It would be of great value to the profession and the public in this country if a scientific investigation were carried out not only of the results of the operation, but also of its physiological effects. An investigation such as this should be attempted by a team of experts at a medical school.

Kux[6] also claims good results in the treatment of asthma. Here, too, the physiological basis of this treatment is not clear. Asthma is notorious for the difficulty in assessing the results of any type of treatment, especially in view of the importance of psychological factors present in asthma. The successes claimed by Kux have been achieved after medical treatment had failed. Other surgeons who have attempted sympathectomy or vagotomy by open operation have in general experienced disappointing results. They have, however, had an occasional success. It is therefore possible that the Kux operation might have a place in the experimental treatment of selected cases that have failed to respond to conservative medical treatment. It will not, however, be possible until some years have passed to arrive at a final assessment of the significance of the operation in cases of asthma.

The Kux procedure is being applied in several other conditions. It is, however, our duty to point out that the procedure is open to abuse, especially because the public often demand the operation on the grounds of symptoms that do not justify its performance. The doctor who indiscriminately carries out this operation for vague abdominal pains and tightness of the chest does not act in the best interests of his patient or his profession.

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3. *Idem* (1953): Dtsch. med. Wschr., **78**, 1590.
4. Kux, E. and Batschwaroff, A. (1954): Thoraxchirurgie, **1**, 377.
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PORPHYRIA IN SWEDEN AND SOUTH AFRICA

GEOFFREY DEAN, M.D., M.R.C.P., *Senior Physician, Provincial Hospital, Port Elizabeth*
and H. D. BARNES, M.Sc., PH.D., A.R.I.C., *South African Institute for Medical Research, Johannesburg*

The clinical and biochemical aspects of the diseases known as the porphyrias, in which disturbances of porphyrin metabolism are associated with a variety of clinical manifestations, have received increasing attention during the past two or three decades. It has been learned that these disorders are not so rare as was formerly believed and that they are, in fact, relatively common in some countries, e.g. Sweden[1] and South Africa.[2] Another important advance has been the elucidation of some of the stages in the biosynthesis of porphyrin pigments from glycine and acetate.[3, 4] From this have come methods for quantitative determination of porphobilinogen and δ-amino-laevulic acid,[5-7] precursors of porphyrins, which are sometimes excreted in quite considerable amounts in the urine. Application of these new methods has made it possible to study cases of porphyria in greater detail.

Early classifications were based on the varied associated clinical manifestations of the porphyrias. Gunther's scheme,[8] translated into modern terminology, provided for 3 groups:

1. Acute porphyria, usually characterized by episodes of acute abdominal pain and vomiting, often accompanied by psychotic manifestations and/or motor paralysis, and the passage of urine which was dark when fresh, or soon became so, and contained an excess of porphyrin. This was later divided into toxic and idiopathic forms but the distinction has lost its significance and has now been abandoned.

2. Chronic porphyria, in which porphyrin excretion and the development of skin lesions on exposed areas began in adult life.

3. Congenital porphyria, in which increased porphyrin excretion and skin lesions began in early childhood. Discolouration of the teeth by deposition of porphyrin was observed in these cases. The skin eruptions were more severe than in the chronic form and adult patients usually showed extensive scarring and even mutilation resulting from recurrent lesions. Congenital porphyria is a very rare condition now known to be essentially different from the chronic form though the two have frequently been

confused because of the marked similarity of their cutaneous manifestations.

In 1937 Waldenstrom[9] reported a large series of cases of acute porphyria in Sweden in whom skin sensitivity did not occur and showed that in addition to porphyrin the urines from these cases also contained porphobilinogen, a colourless substance which could be detected by means of Ehrlich's aldehyde reagent. Porphobilinogen was also found in the urines of patients who had recovered from acute attacks and in small amounts in the urine of some relations who had never suffered from porphyria.[21] A few of the latter later developed acute episodes and the test for porphobilinogen thus provided a valuable means for the detection of some of the latent cases. Waldenstrom proposed a classification of porphyria in which the congenital form remained as defined by Gunther and chronic porphyria was renamed porphyria cutanea tarda because of the late development of porphyrin excretion and photosensitivity, but he suggested that this condition, because of the occurrence of colic, might be related to acute porphyria.

A third classification based on pathogenesis was put forward by Watson *et al.*[10] on studies reported later by Schmid, Schwartz and Watson[11] and Schmid, Schwartz and Sundberg.[12] Congenital porphyria was renamed erythropoietic porphyria because in this type the presence of numerous fluorescent normoblasts in bone-marrow films indicated that the marrow was the site of the metabolic anomaly. Other forms were grouped as hepatic porphyria because excessive amounts of porphyrins or a precursor could be demonstrated in liver tissue from these cases. Though regarded as fundamentally a single entity this group was subdivided according to clinical manifestations into acute intermittent, cutanea tarda and mixed forms of porphyria. Rimington[13] dissents from this view and regards porphyria cutanea tarda as a separate entity and not a mixed form. He and his associates[14] have emphasized that raised stool porphyrins are characteristic of this condition.

Inheritance of Porphyria

Cases of erythropoietic porphyria have occurred in many widely separated countries and multiple occurrence in a sibship has been reported on several occasions. Cockayne[15] discusses the condition as an example of recessive inheritance.

The first indication of the familial incidence of acute porphyria was given by Barker and Estes.[16] While the diagnosis was established in the propositus by spectroscopic examination of her urine this was not done in other members of the family, in whom a provisional diagnosis of porphyria was suggested by clinical manifestations. Gunther[17] pointed out that many cases of porphyria occurred in persons with a peculiar psychological background, which suggested an hereditary constitution (porphyrism) as a basis for the disease. He was unable, however, to find other cases amongst relations of his own patients. The earlier indications for heredity and the difficulties of establishing this possibility are discussed by Waldenstrom,[9] who finally deduced from his extensive material that acute porphyria in Sweden was based on non-sex-linked inheritance of a Mendelian dominant type.

The studies of Dean and Barnes[2] disclosed a similar inheritance for the susceptibility to porphyria in the White population of South Africa and later investigation by Dean (unpublished) traced the inheritance to a pair of early settlers from Holland who married at the Cape in 1688. In these cases, however, symptomatology is mixed, acute attacks occur more frequently in women, cutaneous lesions are commoner and more severe in the men, and some cases show both acute and cutaneous symptoms. This supports Watson's contention that his three subgroups of hepatic porphyria are varied manifestations of the same underlying anomaly, but the findings in Sweden, where acute and cutaneous manifestations of porphyria do not overlap, are not in agreement with this hypothesis.

A comparative study of Swedish and South African cases was clearly indicated, and to this end Dean visited Sweden, where arrangements were made by Waldenstrom for him to see a number of cases, including a family containing several members who had suffered attacks of acute porphyria. Specimens of urine and faeces from these patients and some of their relations were screened for porphyrin metabolites by Dean, quantitative determinations of urinary porphobilinogen and δ-amino-laevulic acid were carried out by Dr. Haegar in Malmö, and the stools were sent to Barnes in Johannesburg for quantitative porphyrin analysis. Similar studies were carried out on two families in Holland with whom contact was provided by Prof. Formyne of the Wilhelmina Gasthuis, Amsterdam. These results and those obtained on excreta from a number of White South African cases are presented and discussed below.

Methods

The methods employed were as follows:

Urine. For screening purposes urine was examined in Wood's light from a portable mercury-vapour lamp for the red fluorescence characteristic of porphyrin. The apparatus described by Harrison[18] was used for spectroscopic detection of porphyrin in varying depths of urine before and after rendering acid to Congo red with hydrochloric acid.

Qualitative examination for porphobilinogen was made by the Watson-Schwartz[19] test with Ehrlich's aldehyde reagent. Porphobilinogen and δ-amino-laevulic acid were determined quantitatively by the procedure of Mauzerall and Granick;[5] amounts up to 1 mg. and 4 mg. per litre respectively were regarded as normal.

Stools. The screening test consisted of examining in Wood's light the extract obtained by rubbing a small fragment of faeces into 2 ml. of a mixture of equal parts of amyl alcohol, glacial acetic acid and ether. Coproporphyrin and protoporphyrin were quantitatively determined by the procedure outlined by Holti *et al.*[20] Coproporphyrin below 15 and protoporphyrin below 45 micrograms per g. dry wt. were regarded as normal. In this study and others somewhat raised stool porphyrins have occasionally been found in

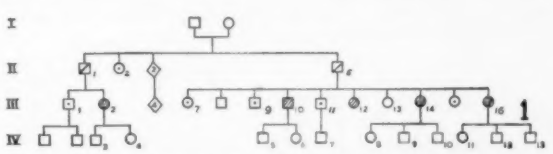

Fig. 1. A Swedish family with many cases of acute porphyria. Squares represent males and circles females. Hatched symbols represent living members with evidence of porphyria, a central dot indicates death with evidence of porphyria and a diagonal bar death without such evidence. See Table I.

TABLE I. (COMPARE FIG. 1)

Member	Acute Attacks	Screen Tests: Faeces porph.	Screen Tests: Urine porph.	Screen Tests: Watson-Schwartz	Urine Analysis: PBG (mg. per litre)	Urine Analysis: ALA (mg. per litre)	Stool Porphyrins: Copro. (μg. per g. dry wt.)	Stool Porphyrins: Proto. (μg. per g. dry wt.)
III/2	1938/52	+	++	++	33	26	22	25
III/10	several mild	neg.	neg.	neg.	2	4	14	15
III/12	mild	neg.	neg.	++	29	12	13	38
III/13	None	neg.	neg.	neg.	1	4	screen test neg.	
III/14	1951	neg.	+	++	42	29	15	24
III/16	1950/56	neg.	+	++	36	23	14	11
IV/3	None	neg.	neg.	neg.	1	4	20	16
IV/4	None	neg.	neg.	neg.			8	28
IV/5	None	neg.	neg.	neg.	1	2	screen test neg.	
IV/6	None	neg.	neg.	neg.	1	4	27	83
IV/7	None	neg.	neg.	neg.	1	4	screen test neg.	
IV/8	None	neg.	neg.	neg.	1	4	screen test neg.	
IV/9*	None	neg.	neg.	neg.	3	4	screen test neg.	
IV/10	None	neg.	neg.	neg.	1	3	screen test neg.	
IV/11	None	neg.	neg.	neg.	1	4	screen test neg.	
IV/12	None	neg.	neg.	neg.	1	4	screen test neg.	
IV/13	None	neg.	neg.	neg.	1	2	screen test neg.	

*The slightly raised urinary porphobilinogen suggests that this might be a latent case.

subjects who had not inherited porphyria, in these cases it is often a temporary phenomenon.

SWEDISH CASES

In this section are included particulars of an affected family and other cases which one of us (G.D.) investigated in Sweden.

The 5 members who had had acute attacks were all in remission when the tests were done, all but one showed pathological increases of porphobilinogen and δ-amino-laevulic acid in their urines, and none had significantly increased stool porphyrins.

Notes on Cases in Fig. 1 and Table I.

II/1 and II/5 were brothers not related to their wives otherwise than by marriage. Although many of their children were porphyrics they do not appear to have died from acute porphyria.

II/2 died in 1910 aged 60. She had suffered from recurrent attacks of abdominal pain. During her final illness she passed red-coloured urine and became paralysed.

III/1 died in 1946 aged 33. He developed severe abdominal pain and had a laparotomy. After the operation there were mental symptoms, he passed red urine, became paralysed and died.

III/2, F.44. She had attacks of acute porphyria in 1938, 1952, 1955 and 1957 after the taking of barbiturates. In some of these she was desperately ill.

III/7. In 1918, aged 20, she became desperately ill, became paralysed and died. She probably had acute porphyria.

III/9. In 1928, aged 30, he complained of abdominal pain and his abdomen was opened, he had fits, passed red urine, became paralysed, and died.

III/10 has had a number of minor attacks, during which he passed red urine.

III/11 died in 1938 aged 32. After taking sedatives he complained of abdominal pain and his abdomen was opened. After the operation he passed red urine, became paralysed, and died.

III/12. Minor attacks only.

III/14 had acute porphyria in 1951 but she made a good recovery.

III/15 died in 1938 aged 22. After taking sedatives she had severe abdominal pain and her abdomen was opened. After the operation she had mental symptoms, passed red urine, became paralysed and died.

III/16. After taking barbiturates in 1950 she developed an acute attack of porphyria. She made a good recovery but had another acute attack in 1956 after taking sleeping tablets.

Notes on Cases in Table Ia

Cases 1 and 1a. This brother and sister are examples of the type of porphyria seen in South Africa. The brother had developed blisters and sores on his hands late in life. Both he and his sister have raised faecal porphyrins. No case of acute porphyria is known as yet in this family.

Case 2. This woman has had a number of acute attacks and when seen still had residual peripheral neuritis. Nevertheless the increase in porphobilinogen was so slight that the Watson-Schwartz test was negative. Her twin sister had died from acute porphyria.

Case 3. This woman is a member of another large porphyric family. Although she was very well when seen, the Watson-Schwartz test was strongly positive.

Case 4. This woman was seen during an acute attack of porphyria. She did not have the marked electrolyte, calcium and potassium loss that usually occurs in South African cases in the acute phase.[27]

No skin lesions were observed in cases 2, 3 and 4. Stool porphyrins were raised in cases 1 and 1a (cutaneous) but not in 2 and 3 (quiescent acute), the increased values for case 4 may be related to the current acute episode. Urinary porphobilinogen and δ-amino-laevulic acid were abnormal in 2, 3 and 4.

While this paper was being prepared similar findings to the above were reported in a number of Swedish patients by Haeger.[26]

DUTCH CASES

In this section are included particulars of two affected families investigated by G.D. in Holland.

TABLE Ia. FINDINGS IN OTHER SWEDISH CASES SEEN BY G.D.

Case and Sex	Screen Tests: Faeces porph.	Screen Tests: Urine porph.	Screen Tests: Watson-Schwartz	Urine Analysis: PBG (mg. per litre)	Urine Analysis: ALA (mg. per litre)	Faeces Analysis: Copro. (μg. per g. dry wt.)	Faeces Analysis: Proto. (μg. per g. dry wt.)
1 M	++	++	neg.	specimen lost		152	55
1a F	++	neg.	neg.	1	2	173	180
2 F	neg.	tr.	neg.	2	7	15	31
3 F	neg.	+	++	19	37	26	25
4 F	+	+	++++	48	20	73	117

TABLE II. (COMPARE FIG. 2)

Member	Acute Attacks	Screen Tests: Faeces porph.	Screen Tests: Urine porph.	Screen Tests: Watson-Schwartz	Urine Analysis*: PBG (mg. per litre)	Urine Analysis*: ALA (mg. per litre)	Stool Porphyrins: Copro. (μg. per g. dry wt.)	Stool Porphyrins: Proto. (μg. per g. dry wt.)
II/1	No	neg.	+++	+		9	53	70
II/2	No	neg.	neg.	neg.			screen test neg.	
II/3	No	neg.	neg.	neg.			screen test neg.	
II/4	Could not be traced for examination or tests.							
II/5	No	?	neg.	neg.			9	13
III/6	Yes	++	+++	++		22	30	115
III/8	No	neg.	neg.	neg.			16	22
III/9	Yes	?	+++	++		16	26	22
IV/4		neg.					screen test neg.	
IV/5		neg.					screen test neg.	

*These urines were analysed by Dr. Haeger about 3 weeks after collection. Porphobilinogen is so unstable that results are not recorded; ALA is more stable and the raised figures recorded are significant.

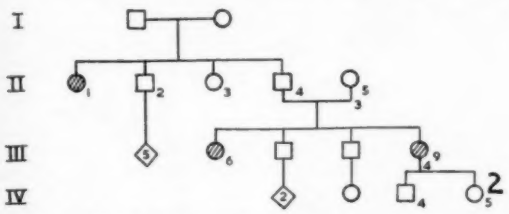

Fig. 2. A Dutch family with 3 cases of porphyria whose clinical manifestations and biochemical findings conform to the pattern seen in Swedish porphyrics. Compare Table II.

Notes on Cases in Fig. 2 *and Table II*

II/1. She has never had a definite attack of acute porphyria but on occasions has noted that her urine was slightly red in colour.

III/6. She has had 4 attacks of acute porphyria, the first when she was 18 years old in 1944 and the last in 1953. Each attack followed the taking of barbiturate sleeping tablets. During the attacks she had severe pain in her abdomen and back and passed red-coloured urine. She had marked weakness in her limbs for some weeks after the attacks. Since 1953 she has stopped all drugs and has remained well.

III/9. In 1951 she became acutely ill with severe pain in her back and legs after taking barbiturate sleeping tablets. She passed red urine and was in hospital for 6 weeks. In 1953 she complained of mild attacks of abdominal pain and underwent a laparotomy under thiopentone anaesthesia. A few days later she developed an acute attack of porphyria with mental symptoms and severe pains in her abdomen, back and limbs. She became paralysed from peripheral neuritis and was in hospital for 3 months. Since then she has taken no drugs and has remained well.

None of the patients in this family showed cutaneous manifestations. Their stool porphyrins are slightly raised but none is as high as is usual in South African porphyric patients.

Notes on Cases in Fig. 3 *and Table III*

II/3. He was a fit man aged 70 years who had never taken sedatives. His skin abraded easily if he knocked the back of his hand but he had no blisters, sores or scars.

II/5. She had suffered from an acute illness and passed dark urine after taking sedatives a few years previously but was otherwise well. The skin on the back of her hands abraded unduly easily.

III/2. She had suffered from recurrent mild attacks of abdominal pain and never felt really well. There were a few depigmented scars on her hands from previous sores and her skin abraded easily.

III/6. The propositus of this family study. He had taken a fair amount of alcohol during 1949, when the skin on the back of his hands started to blister and form small sores. He attributed this to his work as a baker. In 1950 he was in a very nervous state and could not sleep; he took veronal and phenobarbitone

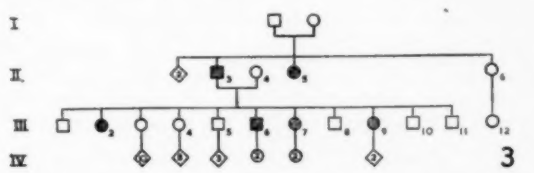

Fig. 3. A Dutch family with several cases of porphyria whose clinical manifestations and biochemical findings conform to the pattern seen in South African porphyrics. See Table III.

TABLE III (COMPARE FIG. 3)

Member	Screen Tests: Faeces porph.	Screen Tests: Urine porph.	Screen Tests: Watson-Schwartz	Stool Porphyrins: Copro. (μg. per g. dry wt.)	Stool Porphyrins: Proto. (μg. per g. dry wt.)
II/3	++	++	neg.	175	263
II/4	neg.	neg.	neg.	screen test negative	
II/5	++	+	neg.	146	192
II/6	neg.	neg.	neg.	screen test negative	
III/2	++	++	neg.	250	485
III/4	neg.	neg.	neg.	17	24
III/5	neg.	neg.	neg.	screen test negative	
III/6	+++	+++	neg.	620	564
III/7	++	+	neg.	730	504*
III/8	neg.	neg.	neg.	screen test negative	
III/9	++	+	neg.	383	645
III/10	neg.	neg.	neg.	45	68
III/11	neg.	neg.	neg.	screen test negative	
III/12	neg.	neg.	neg.	20	65

*This patient had emigrated to Canada and the stool analysis was done for us by Dr. L. A. Brunsting at the Mayo Clinic.

tablets and became acutely ill with mental symptoms and severe pain in his back, abdomen and limbs. His urine was red in colour. In hospital the urine was found to contain porphyrin and porphobilinogen in great excess. All drugs were stopped and he made a good recovery; the Watson-Schwartz test became negative before he left hospital.

III/7. She complains that she cannot sleep, is constipated and has frequent headaches. There has been no attack of acute porphyria but the skin on the back of her hands is unduly sensitive. Dr. Brunsting, of the Mayo Clinic, found high urinary and faecal porphyrin excretion.

III/9. This woman had an acute attack of porphyria in 1952 after taking barbiturates. During the attack the Watson-Schwartz test was positive but it became negative before she left hospital. When seen she was well but the skin on the back of her hands abraded easily.

WHITE SOUTH AFRICAN CASES

Many cases of porphyria have been detected in South Africa. The results in Tables IVa, IVb and IVc were all obtained on White patients of South African stock and those in Table IVd on 2 White patients who had no South African ancestors. The analyses of urine for δ-amino-laevulic acid and porphobilinogen were commenced within 1 hour of collection. A similar metabolic study on Bantu (African) patients is to be published shortly.[23]

Notes on Patients in Tables IVa, IVb, IVc *and* IVd

Case 1 (Table IVa). M28. Porphyria with cutaneous lesions was recognized in 1954. He developed a severe attack of acute porphyria in 1957. The results recorded were obtained on admission to hospital and again 5 weeks later when considerably improved but still far from recovered.

Case 2 (Table IVc). F35. Her skin has abraded easily for as long as she can remember but she has had no outspoken acute attack.

Case 3 (Table IVc). F41. Two attacks of severe abdominal pain with prostration were probably episodes of acute porphyria though not diagnosed at the time. She observed fragility of her skin during pregnancy but not at other times.

Case 4 (Table IVc). M54. Has never had acute porphyria but his skin was somewhat fragile between his 20th and 40th years.

TABLE IVa. SOUTH AFRICAN PATIENTS DURING ACUTE EPISODES

Case No.	Acute Attacks	Urine: Porphyrin	Urine: Watson-Schwartz	Urine: PBG (mg. per litre)	Urine: ALA (mg. per litre)	Stool Porphyrins: Copro. (μg. per g. dry wt.)	Stool Porphyrins: Proto. (μg. per g. dry wt.)
1.	Current	+++	+++	194	219	890	1,400
	5 wks. later	tr.	neg.	2	7	270	508
6.	Current	++	+	25	26	620	1,240
10.	Current	+	+++	99	45	not received	
11.	Current	tr.	++	40	41	805	810
13.	Current	+	++	138	81	623	805
14.	Recent	+	+	20	8	345	568
17.	Current	tr.	++	31	30	670	785

TABLE IVb. SOUTH AFRICAN PATIENTS IN THE SUB-ACUTE PHASE

Case No.	Acute Attacks	Urine: Porphyrin	Urine: Watson-Schwartz	Urine: PBG (mg. per litre)	Urine: ALA (mg. per litre)	Stool Porphyrins: Copro. (μg. per g. dry wt.)	Stool Porphyrins: Proto. (μg. per g. dry wt.)
7.	Subacute	tr.	neg.	3	3	363	266
8.	Subacute	tr.	neg.	8	5	477	930
13.	Recent	tr.	neg.	6	7	623	392
17.	Recent	+	?+*	24	81	580	890
20.	Subacute	tr.	neg.	6	11	630	760

*This urine contained a large amount of urobilinogen, which complicated the interpretation of the Watson-Schwartz test.

TABLE IVc. SOUTH AFRICAN PATIENTS, LATENT OR IN REMISSION

Case No.	Acute Attacks	Urine: Porphyrin	Urine: Watson-Schwartz	Urine: PBG (mg. per litre)	Urine: ALA (mg. per litre)	Stool Porphyrins: Copro. (μg. per g. dry wt.)	Stool Porphyrins: Proto. (μg. per g. dry wt.)
2	None	tr.	neg.	2	2	1,220	1,310
3	1940/41	neg.	neg.	1	2	54	145
4	None	neg.	neg.	1	5	106	186
5	None	neg.	neg.	0	1	70	165
9	1954	tr.	neg.	2	3	414	490
9a.	1953	tr.	neg.	2	5	315	348
10	Recent	neg.	?*	2	3	520	574
10a.		tr.	neg.	3	8	126	197
12	1957	tr.	?+	3	1	593	577
18	None	tr.	neg.	1	1	743	2,000
19	None	tr.	neg.	1	2	159	131

* This urine contained pyridium, which complicated the reading of the Watson-Schwartz test.

TABLE IVd. SOUTH AFRICAN PATIENTS WITH NO SOUTH AFRICAN ANCESTORS, IN REMISSION.

Case No.	Acute Attacks	Urine: Porphyrin	Urine: Watson-Schwartz	Urine: PBG (mg. per litre)	Urine: ALA (mg. per litre)	Stool Porphyrins: Copro. (μg. per g. dry wt.)	Stool Porphyrins: Proto. (μg. per g. dry wt.)
15	1945	++	++	76	47	36	64
16	1954/5	+++	+++	176	80	44	131

Case 5 (Table IVc). F. Traces of porphyrin were detected in urine in 1954 and stool contained 395 μg. of coproporphyrin and 740 μg. of protoporphyrin per g. dry wt. She has never had acute porphyria and the only indication of skin fragility was easy formation of blisters at the base of a thumb when playing tennis.

Cases 1, 2 and 3 are siblings and 4 and 5 are father and daughter.

Case 6 (Table IVa). F43. Had had cutaneous eruptions on exposed areas for the past 8 years. Scarring was marked and some active lesions were present. There had been bouts of intermittent epigastric discomfort, which had recently become worse. Gallbladder trouble was suspected but the findings recorded indicate mild acute porphyria.

Case 7 (Table IVb). M52. Porphyria with cutaneous lesions was recognized in 1954. Recent headaches and abdominal pain had largely subsided when the results recorded were obtained but suggest a subacute porphyric episode.

Case 8 (Table IVb). F39. Porphyria was first diagnosed in 1948 on admission to a neuropsychiatric hospital for investigation. She has had a recurrent rash of an eczematoid nature for some years rather than the characteristic bullous dermatosis. During exacerbations she feels weak and tired and headaches are sometimes severe. The results recorded were obtained on one of these occasions. She had no pain at the time but the findings suggest a subacute phase.

Case 9 (Table IVc). M29. There have been several mild recurrences of abdominal pain since his attack of acute porphyria. His skin is fragile and scars are present on exposed surfaces. When examined he had been free from acute symptoms for some years and was living an active life, participating in vigorous sport at week-ends.

Case 9a (Table IVc). F57. In 1953 a cold was treated with sulphadiazine. Abdominal pain occurred and a hysterectomy was performed. Paralysis of legs and arms developed within a month and lasted for about a year. Porphyria was not suspected, but after her son (case 9) had his acute attack examination of urine and faeces disclosed the presence of excessive amounts of porphyrin. No scarring was apparent on her arms or face.

Case 10 (Tables IVa and IVc). F47. This patient has never enjoyed good health and was frequently given sedatives by her doctors. From July 1956 a phenobarbitone preparation was used with no apparent ill-effects. She suffered a grave psychological trauma in August 1957 and soon afterwards acute porphyria commenced with gross peripheral neuritis. No cutaneous manifestations were observed. The results in Table IVa were obtained during the acute episode and those in IVc 3 months later, when the only residual sign was a mild contracture of the fingers of one hand.

Case 10a (Table IVc). F. Is a daughter of case 10. She accompanied her mother on the recent visit to the consulting physician for follow-up examination. Though not making any complaints at the time she followed the suggestion to send specimens for examination; these gave the results recorded.

Case 11 (Table IVa). M. Two laparotomies in the preceding 3 months afforded no relief of pain, constipation and sleeplessness. Porphyria was detected in time to prevent a third exploration. This patient is an exception to the common finding in South African male porphyrics in suffering an acute attack while presenting no evidence of skin lesions. He stated that a sister was troubled by cutaneous eruptions.

Case 12 (Table IVc). M31. Typical skin lesions have been present for many years. Meningitis in March 1957 was followed by acute porphyria with paralysis 3 months later. When the results recorded were obtained in January 1958 he complained only of slight weakness of the muscles of his right hand.

Case 13 (Tables IVa and IVb). F35. Ectopic pregnancy was suspected but the physical signs were inconclusive. Dark urine contained porphyrin and porphobilinogen. Hyperpigmentation and facial hirsuties were noted and she stated that her skin abraded easily. Three months elapsed between the two sets of analysis.

Case 14 (Table IVa). F. Symptoms attributable to acute porphyria began while she was on holiday; after she returned home porphyrin was detected in her urine but the porphobilinogen reaction was inconclusive because the specimen had been in transit for 48 hours. Three weeks later she was able to make the 200 mile journey to Johannesburg without difficulty and the results recorded were obtained. She then had a blister on one hand and showed numerous scars of previous lesions which began during pregnancy 9 years previously.

Case 15 (Table IVd). F54. This woman was first seen during an acute porphyric episode in 1945 and has fully recovered from the residual paresis of her left arm. There has been no recurrence of symptoms, even during an attack of infective hepatitis in 1952. There is no history or evidence of the cutaneous manifestations associated with porphyria, but she has complained of an intense irritation of the skin of her forearms during high summer.

Case 16 (Table IVd). F31. She is a daughter of case 15, has been hospitalized for 2 severe attacks of acute porphyria, and has had a third minor episode. She has never had any skin trouble.

Cases 15 and 16 are of British stock and have no South African ancestors. The clinical manifestations and biochemical findings conform to the pattern found in acute porphyria seen in Sweden.

Case 17 (Tables IVa and IVb). M29. Fragility of his skin was first noticed 8 years ago following burns of the forearms with hot porridge. He now has scarred forearms and hands. Temporal hirsutism was noted but there have been no lesions on his face. A tired feeling in his legs was followed by acute epigastric pain for which he was referred to a surgeon; the results in Table IVa were obtained at the time. Five months later, when apparently well, he came for follow-up tests (Table IVb). Porphobilinogen and δ-amino-laevulic acid were much higher than those of other patients in the same group and the urine contained a marked excess of urobilinogen. Further examinations will be carried out as opportunity presents.

Case 18 (Table IVc). F33. Her hands began to blister 7 years ago when they were subjected to frequent trauma during handling of motor spares. The lesions have been more or less continuous since and have spread to her arms, face, V-area of her chest, and ankles, which were badly scarred. Considerable facial hirsutism was noted. There was no history of neuritic or abdominal pains.

Case 19 (Table IVc). M49. Blisters following negligible trauma to hands began about 15 months ago and have recurred frequently since. He has had no abdominal pain or operations.

Case 20 (Table IVb). M23. For two or three years he has had lesions on his hands which commence as spontaneous blisters; he does not relate them to trauma. He has had no operations but states that when many lesions are present he loses appetite, experiences abdominal pain, and vomits after taking fatty food. This story and the slightly increased porphobilinogen and δ-amino-laevulic acid suggested the possibility of a smouldering sub-acute phase.

DISCUSSION

The results from this study given in detail in Tables I, Ia, IVa, IVb, IVc and IVd are summarized in Table V. These show that the differing clinical manifestations of porphyria in Sweden and South Africa are paralleled by differences in biochemical findings. The opportunity to study the condition in these two countries has been most instructive, for the differences stand out in high relief though some features are common to both. The similar mode of inheritance in the two countries has already been mentioned. Because the differences discussed below breed true in the two communities it is believed that the diseases depend on anomalies in different genes and are not varied manifestations of one underlying disorder. The occurrence of acute and cutaneous manifestations in separate families in Sweden and in the same family in South Africa does not support an earlier suggestion[2] that differences in climatic conditions such as the amount of solar irradiation play a significant role. When these genetic forms occur in the same community extensive investigation may be required to determine in which of the two any given patient belongs. The findings in the Dutch families (Figs. 2 and 3, Tables II and III) show that separation can be achieved.

As will be shown below the form of porphyria seen in the White South African patients does not fit comfortably into the existing classifications, and the name variegate porphyria (porphyria variegata) is proposed for the genetic disorder of pyrrole-porphyrin metabolism characterized clinically by acute attacks or cutaneous symptoms and not infrequently

TABLE V. SUMMARY OF FINDINGS IN TABLES I, Ia, IVa, IVb, IVc AND IVd

	No. of Cases	Urine: Porphyrin	Urine: Watson-Schwartz	Urine: PBG (mg. per litre)	Urine: ALA (mg. per litre)	Stool Porphyrins: Copro. (μg. per g. dry wt.)	Stool Porphyrins: Proto. (μg. per g. dry wt.)
Normal		neg.	neg.	less than 1	less than 4	0-15	0-45
Sweden							
Acute phase	1	+	+++	48	20	73	117
Remission	7	neg. to +	neg. to ++	2-42 (21)	4-37 (20)	13-26 (17)	11-38 (24)
Cutaneous cases	2	neg., ++	neg.	1	2	152; 73	55; 180
South Africa							
Acute phase	7	tr. to +++	+ to +++	20-194 (78)	8-219 (64)	345-890 (659)	568-1,400 (935)
Sub-acute	5	? to +	neg. to +	3-24 (9)	3-81 (21)	363-630 (535)	266-930 (648)
Latent and remission	11	neg. to tr.	neg to ?	0-3 (1·6)	1-8 (3·0)	54-1,220 (393)	131-2,000 (557)
Imported cases in remission	2	++, +++	++, +++	76; 176	47; 80	36; 44	64; 131

Watson-Schwartz = qualitative test for porphobilinogen
PBG = Porphobilinogen ALA = δ-amino-laevulic acid
Grossly pathological values are enclosed in rectangular frames.

by both. Acute porphyria, it is suggested, should cease to be the name for a clinical entity and should be restricted, in a descriptive sense, to the acute episodes of abdominal pain, neurological disorder or psychiatric disturbances which happen to constitute the main clinical picture of intermittent acute porphyria but are also a feature in many cases of variegate porphyria. Dissatisfaction has been expressed for the name intermittent acute porphyria on the grounds that the primary disturbance is at the precursor rather than the porphyrin stage and the occurrence of similar acute episodes in variegate porphyria makes for more confusion and renders it still less suitable. The name, is however, retained for the present to denote the condition exemplified by the Swedish group in which cutaneous lesions do not occur. These disorders are not restricted to the countries in which our cases have been found and it is hoped that study along the lines indicated will enable cases found elsewhere to be classified more exactly than has hitherto been possible.

In many respects porphyria variegata corresponds with the form known as porphyria cutanea tarda which Rimington and his associates[14] regard as a separate entity and not as mixed forms of the disease. For the condition seen in South Africa, however, porphyria cutanea tarda is an inapt name for two reasons: (*a*) a number of patients suffer acute episodes but present no cutaneous lesions whatever, and (*b*) some patients are known in whom skin lesions began in early childhood[22] and cannot, therefore, be regarded as delayed. The term mixed porphyria proposed by Watson is not a suitable alternative for variegate porphyria, because it is now clear that in this condition the varied clinical manifestations arise in patients who have inherited a single genetically determined constitutional anomaly, which is, however, different from the anomaly inherited in intermittent acute porphyria.

For some years it has been known that urinary porphobilinogen is increased during acute episodes in both intermittent acute and variegate porphyria. More recently it has been learned that this is also true of δ-amino-laevulic acid. The findings summarized in Table V show that, while excretion of these substances remains abnormal (sometimes for years) in patients in remission from intermittent acute porphyria, it returns to normal or nearly so during recovery from the acute episode in patients with variegate porphyria. The Watson-Schwartz test thus provides a valuable means for detecting quiescent cases of the former but is useless for this purpose in the latter since fresh specimens of urine consistently give negative results when acute symptoms are absent.

The outstanding constant feature of variegate porphyria is the increased excretion of copro- and protoporphyrin in the faeces. This is often marked irrespective of past or present acute episodes and has been observed[23] in some members of affected families, including young children, who have never presented either acute or cutaneous symptoms. Faecal excretion of these porphyrins by patients with intermittent acute porphyria, on the other hand, is almost always within normal limits. The simple screening procedure for stool porphyrins not only provides a method for detecting latent or quiescent variegate porphyria but also serves to distinguish this from the intermittent acute form. It must, however, be emphasized that increase in stool porphyrins, whilst strongly suggestive of porphyria when found in a member of a family known to be affected, cannot always be thus interpreted.[24]

Our experience in this connection has been limited to but one case of intermittent acute porphyria in an acute phase, who showed a slight increase in stool porphyrins. In this condition the fundamental metabolic disorder is the escape of precursors from the intracellular enzyme system responsible for their transformation into porphyrinogens. On these grounds no great increase in production and

excretion of these pigments would be expected. It is also pointed out that diminished faecal solids, resulting from reduced food intake during acute episodes, would lead to a slight increase of porphyrins when expressed in terms of dry or wet stool.

These biochemical findings indicate that in variegate porphyria the fundamental disturbance of porphyrin biosynthesis occurs at a later stage than in intermittent acute porphyria.[25] The precursors are satisfactorily transformed to the stage corresponding to copro- and protoporphyrin and only then escape from the intracellular enzyme system. This system is, however, sensitive to incidents such as the use of certain drugs which interfere at the δ-amino-laevulic acid and porphobilinogen stage and precipitate typical attacks of acute porphyria. It is strongly recommended that a conservative attitude should be adopted in the administration of drugs to patients in whom there is a possibility of porphyria and that barbiturates and sulphonamides should not be prescribed for them.

SUMMARY

Quantitative determinations of δ-amino-laevulic acid and porphobilinogen in urine and copro- and protoporphyrin in faeces have been carried out on excreta from porphyric patients in Holland and Sweden and amongst White members of the population of South Africa.

The results, which are discussed in relation to the clinical manifestations of the associated diseases, indicate that intermittent acute porphyria is a different genetic disorder from the condition which presents with acute episodes or cutaneous lesions and sometimes with both. The name variegate porphyria (porphyria variegata) is proposed for the latter.

The main features whereby these two forms can be differentiated are:

1. Cutaneous manifestations, which are common in variegate porphyria, are never seen in affected members of families with the intermittent acute form.

2. In the latter, excretion of porphobilinogen and δ-amino-laevulic acid continues at a high level, sometimes for years, after the attack has subsided, but in variegate porphyria it returns to normal or virtually normal levels as the patient recovers.

3. Raised stool copro- and protoporphyrin are characteristic of variegate porphyria even during remission from acute episodes and of latent cases without clinical manifestations. In intermittent acute porphyria stool porphyrins may be slightly raised during the acute attack but are otherwise normal or nearly so.

This comparative study would not have been possible without the kind cooperation of Prof. Jan Waldenstrom, who gave access to the Swedish patients, and Dr. B. Haeger, who carried out the determinations of δ-amino-laevulic acid and porphobilinogen in the urines of the Swedish and Dutch patients. We are indebted to Prof. P. Formyne, of the Wilhelmina Gasthuis, Amsterdam, for contact with the two Dutch families, and to Dr. L. A. Brunsting for analysis of the specimens from a member who had emigrated to Canada. Many doctors in South Africa assisted in this project. Dr. E. H. Cluver, Director of the South African Institute for Medical Research, granted facilities for biochemical analysis and Miss C. E. Campbell prepared drawings and photographs of the family trees. The name porphyria variegata was chosen from suggestions made by Prof. S. Davis, Department of Classics, University of the Witwatersrand. The South African Council for Scientific and Industrial Research made a grant-in-aid to Dr. Geoffrey Dean for comparative studies in South Africa and overseas.

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'THE NATURAL SHOCKS THAT FLESH IS HEIR TO'

DOSTOEVSKY ON HIS MELANCHOLY *

I gradually began at dusk to sink into that condition which is so common with me now at night in my illness, and which I call *mysterious horror*. It is a most oppressive, agonizing state of terror of something I don't know how to define, and something passing all understanding and outside the natural order of things, which yet may take shape this very minute, as though in mockery of all the conclusions of reason, come to me and stand before me as an undeniable fact, hideous, horrible, and relentless. This fear usually becomes more and more acute, in spite of all the protests of reason, so much so that although the mind sometimes is of exceptional clarity at such moments, it loses all power of resistance. It is unheeded, it becomes useless, and this inward division intensifies the agony of suspense. It seems to me something like the anguish of people who are afraid of the dead. But in my distress the indefiniteness of the apprehension makes my suffering even more acute.

* *The Insulted and the Injured.* By Fyodor Dostoevsky.

GASTRECTOMY*

J. WOLFOWITZ, M.B., F.R.C.S. (EDIN.)

President, Southern Transvaal Branch, Medical Association of South Africa, 1958

The reason for the choice of gastrectomy as the subject of my discourse tonight is that, despite the fact that this operation has become a common and popular procedure, its history is nevertheless a recent one. It is a procedure that merits meticulous attention to detail in the choice of suitable candidates and the performance of the operation as well as the pre- and post-operative treatment, in order to achieve the excellent results that follow upon this procedure when adequately and efficiently performed.

Dr. Wolfowitz
'*Portrait Moderne*' *by Jane Plotz*

The history of gastrectomy is of extreme interest. In the year 1929 Finney and Rienhoff,[1] in an exhaustive review of gastrectomy, found 67 cases in the world literature. This was only 30 years ago, and today that figure is the number of gastrectomies performed by an average surgeon in one year of practice. This indicates the tremendous progress this procedure has made. These authors stated: 'It would appear that credit for having first conceived the idea of resection operations on the stomach must be given to a certain famous professor, highly respected and renowned amongst the medical profession of Philadelphia.'[1] This statement is based on the authority of Merren, of Giessen, who in 1810 contributed a monograph entitled 'Certain surgical operations and experiments on animals, illustrated by facts'. The surgeon's name was not mentioned, nor has it ever been discovered. It appears that the operation was conceived by this unknown surgeon out of the sufferings of a colleague and beloved friend of his, a certain Dr. Middleton. The hazard was so great in the eyes of this far-thinking surgeon that he would not undertake the operation on his friend, but 2 years later performed it on several dogs, resecting the pylorus—in every case with a fatal termination.

The study of the data and a comparison of dates led others to believe that this surgeon was John Jones, a native of Philadelphia, who was the first Professor of Surgery at Kings College, New York, subsequently to become the College of Physicians and Surgeons, and now known as Columbia University.

Merren, stimulated by this work, continued the experiments on dogs, and concluded that the operation, although a very difficult one, was feasible for humans. His criteria for surgery enunciated nearly a century and a half ago would be acceptable even today. They were, in his own words:

1. If the patient seem a sure prey of death, after having been sick for a long time, and after every other attempt had been tried to no avail;
2. If we find by placing the fingers within the right region an unmistakable hardening;
3. If a short time after eating the patient suffers from obstruction of the bowels and chronic vomiting.

In 1877 Billroth made his famous prophecy whilst speaking on the operation of gastrorrhaphy, i.e. the closing of a fistulous opening in the stomach: 'From this operation to the resection of a piece of carcinomatous stomach there is still only a bold step to be taken.' In 1881, only 4 years later, he performed the first pylorectomy for carcinoma. In 1884 Phineus Connor, of Cincinatti, performed the first total gastrectomy in man, but the patient, moribund at the time of operation, died on the table of shock.

In 1897 Schlatter performed the first successful total gastrectomy in man, anastomosing the oesophagus to the jejunum. This patient, a woman aged 56, survived for 1 year and 53 days, when she died of a recurrence of her carcinoma. Brigham, of Boston, reported the second successful case in 1898, and anastomosed the oesophagus to the duodenum. Richardson, of Boston, in the same year reported the third, and in 1900 Marvil reported the fourth.

Patterson, in a Hunterian lecture in 1906, reported on a total of 27 total gastrectomies in the world literature. Since this lecture the advance of gastric surgery has been rapid, and has included the illustrious names of Finsterrer, Mayo Robson, the Mayo brothers, Polya, Balfour, Hofmeister, Moynihan, and a host of other legendary figures who pioneered this work and reduced the mortality from 53·8% in 1929 to 1% today.

It is noteworthy that this procedure originated in a desire to alleviate suffering that responded to no other form of known treatment, or in an attempt to save life where other treatment had failed. These basic indications remain the indications for surgery of the stomach today, and I therefore introduce the subject of gastrectomy with the indications for its use.

INDICATIONS FOR GASTRECTOMY

A. Carcinoma

This is the prime indication for this type of surgery, and gastrectomy remains the only treatment today for carcinoma of the stomach, the duodenum (first and second parts)—in association with other surgery for carcinoma of the ampulla of Vater—the pancreas, and the lower end of the oesophagus. The value of total gastrectomy for carcinoma of the stomach has been open to criticism in recent years. If the lesion is such as to warrant so extensive a procedure as total gastrectomy plus splenectomy plus removal of the omentum plus a gland clearance, then obviously it is not the correct answer to this disease. The success of the manœuvre is debatable because it only prolongs life for a short while, and attendant upon this type of surgery is the post-operative life of intestinal invalidism marked by diarrhoea, nutritional disturbance, haematopoietic deficiency, vitamin insufficiency, inanition, and even possible eventual death from the physiological upset caused by the operation, or death from recurrence of a disease that was so extensive as to warrant such a radical undertaking.

It is my opinion, therefore, that the indications for total gastrectomy are small, and will dwindle still more in the future.

Partial gastrectomy, however, remains today the only available method of treatment for malignant disease of the stomach, the lower end of the oesophagus and the first part of the duodenum. Physiologically this also is not the final answer in the treatment of the disease, but until other weapons become available, it will remain the only resource in the attempt to cure the cancer, prolong life, or alleviate suffering. There is a wide concept in the profession that carcinoma of the stomach is still to be regarded as a fatal disease despite surgery, but this concept must be denied, for it is the experience of many of us present here this evening that carcinoma of the stomach, the lower end of the oesophagus, the duodenum and the ampulla of Vater can be cured by subtotal gastrectomy. For carcinoma of the stomach my own 5-year survival rate is 20%; accepted figures all over the world vary from 19 to 25%, but in one or two centres the rate is given as high as 30%.

Since publishing my original article on total gastrectomy in December 1950 I have been unhappy about the procedure, and latterly have almost entirely abandoned the total operation. Pack, of New York,[2] has recently stated that carcinomas of the stomach, treated by subtotal gastrectomy in 95% of cases, will be cured if recurrence has not manifested itself within 3 years of the date of operation.

B. Sarcoma

Sarcomas of the stomach comprise only 1% of all tumours of the stomach. They may be leiomyosarcoma, spindle-cell sarcoma, lymphosarcoma, neurogenic sarcoma, and myeloid infiltration of the stomach; 40% of sarcomas of the stomach are lymphosarcomas. The diagnosis of sarcoma of the stomach is only established with any certainty by histology. Gastrectomy is the treatment of choice in this tumour, although in some cases the tumour is sensitive to radiation and cure or relief by radiotherapy is possible.

* Valedictory Presidential Address, Johannesburg, 17 February 1959.

C. Benign Tumours

Adenoma is the commonest type of simple tumour of the stomach and, because of its tendency to undergo malignant change, partial gastrectomy is the treatment for this condition. Because of the difficulty of macroscopic differentiation it would appear that other simple tumours of the stomach warrant a similar form of treatment, these being leiomyomas, fibromyomas, adenomyomas, myxofibromas, haemangiomas, fibromas, lipomas, neurofibromas, and dermoid cysts.

Should the diagnosis be established in these tumours without doubt by frozen section performed at the time of operation, then, in adenoma of the stomach, sleeve resection of the portion of the stomach wall involved is permissible in the isolated tumours, but more extensive resection is advocated for multiple simple tumours, even to the extent of total gastrectomy for polyposis of the stomach.

D. Syphilis

Syphilis of the stomach, listed in many previous text-books of surgery as a disease of the stomach that may warrant surgery, has been omitted in the more recent publications. Shackelford, in his *Surgery of the Alimentary Tract* published in 1955, omits the disease entirely.

In 25 years of practice I have come across only one case of a gumma of the stomach diagnosed pre-operatively, in a man with a long syphilitic history in whom, despite exhaustive antisyphilitic treatment, no response was forthcoming. Gastrectomy was performed for relief of symptoms, which was complete.

E. Volvulus

Volvulus of the stomach is not a common condition, but I have had the privilege of dealing with 6 cases. There are 2 types of volvulus, viz. the organo-axial and the mesentero-axial, indicating the axis round which the volvulus occurs. Volvulus of the stomach may be primary or secondary. In the secondary type it is associated with such conditions as carcinoma of the transverse colon, enlarged spleen, and simple or malignant tumour of the stomach or pancreas. In the primary type the cause is not definitely known; it is found in the obese individual with a heavy pendulous omentum as well as a large 'J'-shaped stomach, despite a broad costal margin.

In my opinion the treatment of choice for volvulus of the stomach is a partial gastrectomy with removal of the omentum. There are, however, other schools of thought; for instance, Norman Tanner advises fixation of the stomach to the under surface of the liver, with a shortening of the gastro-hepatic omentum. The results of gastrectomy are so good in this condition that I unhesitatingly advise it.

F. Trauma of the Stomach

Trauma of the stomach is not unusual, but on the occasions that I have seen this condition only once was it necessary to perform a gastrectomy because of the massive destruction of the stomach. Usually the lesion can be repaired.

G. Corrosive Poisons

A further indication for surgery of the stomach is the late effects of corrosive poisons with residual deformities of the stomach and possible obstruction.

H. Chronic Hypertrophic Gastritis

I have on several occasions had to perform gastrectomy for hypertrophic gastritis where this has been associated with prolapse of the gastric mucosa into the duodenum or with haemorrhage. Patients suffering from this condition are often disabled with severe dyspepsia or multiple haemorrhage, which are not easily relieved by medical measures.

I. PEPTIC ULCER

The vexed question of the peptic ulcer should be separately considered under duodenal ulcer and gastric ulcer, for the reason that duodenal ulcer is primarily a concern of the physician, whereas gastric ulcer should be considered as a surgical disease. I have laid down the principle in my own practice that all gastric ulcers, particularly acute ones, when first discovered, should be given 4 weeks of stringent medical treatment with hospitalization. If at the end of the 4 weeks the patient has not completely recovered, both clinically and radiologically, then I advise surgery. There are, however, further criteria which indicate immediate surgery rather than a 4 weeks' trial of medical treatment, viz. (*a*) gastric ulcer with radiological evidence of a large crater, (*b*) gastric ulcer with achlorhydria, (*c*) a gastric ulcer of any description on the greater curvature, and (*d*) chronic pre-pyloric gastric ulcer.

In duodenal ulcer, surgery is indicated in the following conditions:

1. *Perforation.* The operation may be either closure or gastrectomy, depending on the circumstances of each individual case. In a survey of 2,224 cases from 16 Scandinavian hospitals Andreas Hoyer,[2] of Oslo, reports that in 8 hospitals immediate partial gastrectomy was performed for perforated peptic (gastric or duodenal) ulcer; in 5 hospitals only closure of the perforation; and in 3 hospitals there was no standard treatment. In the 2,224 cases the percentage of duodenal ulcers was 72% (Table I).

TABLE I. TREATMENT OF 2,224 PATIENTS WITH PERFORATING GASTRIC OR DUODENAL ULCERS IN 16 SCANDINAVIAN HOSPITALS (ANDREAS HOYER[3])

Treatment	*No. of Cases*	*No. of Deaths*	*Rate of Mortality %*
Simple suture or excision and suture	1,364	137	10·0
Partial gastrectomy ..	763	43	5·6
Conservative treatment (no operation)	97	49	50·5
Total	2,224	229	10·3

2. *Pyloric obstruction* or hour-glass constriction of the stomach.

3. *Haemorrhage.* Operation for haemorrhage in gastric and dudodenal ulcer is attended by a total mortality of 20%, according to a report by Snyder and Berne[4] of the University of Southern California. They state that the mortality of haemorrhage from gastric ulcer not operated on was 75%, and of haemorrhage from duodenal ulcer not operated on, 12·3%. This is a strong argument in favour of treating haemorrhage from a gastric ulcer surgically. My own mortality generally for peptic ulcer is 10% when surgery is performed for haemorrhage.

Acute haemorrhage occurring under the age of 40 from duodenal ulcers should be treated conservatively, because in the large majority of cases the haemorrhage ceases in 24-36 hours. With gastric ulcers this may not be the case, and a close observation should be maintained for 36 hours. If haemorrhage persists after 36 hours, as evidenced by a rising pulse rate or drop in blood pressure and a drop in haemoglobin plus repeated haematemesis or melaena, it is then my practice to institute surgery immediately for either gastric or duodenal ulcers.

In patients over the age of 40 the likelihood that haemorrhage will cease of its own accord drops progressively with age because of the state of the arterial tree. If the patient recovers from an initial haemorrhage I apply the dictum that no patient with a peptic ulcer should be allowed to bleed more than twice and therefore two episodes of haemorrhage are an indication for surgery.

4. *Failure* of reasonable and adequate *medical therapy* is an indication for surgery in duodenal ulcer. By reasonable and adequate medical therapy I imply that hospitalization and correct medication and diet have been tried on at least two independent occasions with symptomatic and radiological improvement in the ulcers. A practitioner does not hesitate to advise surgery for other types of pain. Why then subject the patient with duodenal ulcer to years of episodes of pain and still maintain him on medical treatment?

5. *Economic factors.* With the hastening trend of modern living it is necessary for the breadwinner to be fit and well in order to maintain his economic and social position. When he is incapacitated for a long time from duodenal ulcers, with intervals of time in bed or off work, the economic factor becomes one of the commonest indications for surgery.

6. *Penetration.* The duodenal ulcer that is penetrating because of chronicity, with pain in the back, etc.

7. *Blood group.* Where the patient belongs to a difficult blood group.

Surgical Procedures

The national committee on surgical procedures for peptic ulcer of the American Gastroenterological Association have evaluated the results of various surgical procedures undertaken in 1,923

cases of duodenal ulcer. For gastric ulcer it is accepted that partial gastrectomy alone is the operation of choice, but for duodenal ulcer the procedures advised were (1) vagotomy, (2) gastro-enterostomy, (3) vagotomy plus gastro-enterostomy or pyloroplasty, and (4) partial gastrectomy. The results of vagotomy were indifferent. Gastro-enterostomy has been abandoned except in the case of complete pyloric obstruction in a patient who is desperately ill. After vagotomy plus gastro-enterostomy the incidence of stomal ulcers was much higher than with gastrectomy, and haemorrhage still occurred from the original duodenal ulcer. The conclusion of the committee, therefore, is that partial gastrectomy is the operation of choice in duodenal ulcer.

Results of Partial Gastrectomy

The mortality in my cases of gastrectomy, excluding those operated on for haemorrhage, is 1·2% (6 deaths), but in those operated on for haemorrhage the mortality is 10%.

The average length of stay in the nursing home is 2 days for pre-operative preparation, and 11 days for post-operative care.

The immediate complications are as follows:

Subphrenic abscess (2 cases)	0·4%
Haemorrhage requiring operation	*Nil*
Haemorrhage requiring conservative treatment (1 case)	0·2%
Intestinal obstruction requiring operation (2 cases)	0·4%
Duodenal stump leakage	0·4%
Temporary oedema of the stoma	10·0%
Peritonitis	*Nil*
Pancreatitis	*Nil*

The delayed complications are as follows:

Stomal ulcer	*Nil*
Dumping syndrome—immediate	10·0%
after 6 months	5·0%
after 1 year	1·0%
Afferent loop syndrome	*Nil*
Post-gastrectomy inanition (3 cases)	0·6%

These figures are accurate as far as can be ascertained but, should stomal ulcer have occurred in cases that I have operated on, it is possible that they may have gone elsewhere for further treatment.

Burger and Pick,[5] in a study of 301 patients with duodenal ulceration treated by means of vagotomy and gastro-enterostomy, report stomal ulceration or recurrent ulceration in 4·25% of cases. The same authors report the incidence of malignancy in gastric ulcer as 15%.

Santy, Michaud and Garde,[6] in reviewing 102 recurrent ulcers after gastro-enterostomy and 23 after gastrectomy, report an incidence of 15-20% of recurrent ulceration after gastro-enterostomy and 3 to 4% after gastrectomy. In most of these the recurrent ulceration occurred in the first 2 years.

It would appear from these results that the treatment of choice for peptic ulceration is partial gastrectomy; but at this stage it should be stated that if the incidence of stomal ulcer is to be kept low, then the gastrectomy must be high; in other words, 7/8ths of the stomach must be removed to ensure that the whole acid-bearing area of the stomach has been ablated.

PROCEDURE

Gastrectomy has become so common that there is a tendency amongst surgeons to treat the operation lightly, and this will lead to mortality, failure, and poor results generally. The operation is one of some magnitude, and must always remain so, and it requires to be treated with the respect it deserves. To that end I have laid down the following principles:

1. Gastric surgery should not be undertaken by a lone surgeon. If the results are to be adequate, like most major surgical procedures it requires the services of a team, which should consist of the surgeon and a permanent assistant such as a partner, or a full-time assistant, or a registrar who regularly works with the surgeon. Every surgeon doing this type of surgery should have his own theatre sister, who becomes an expert in the technique of the operation. The ward nursing staff must be constant, and must periodically be instructed in the pre- and post-operative care of these patients so that the regime laid down shall be accurately followed. The surgeon should regularly employ the same anaesthetist, who in turn will accustom himself to the blood and fluid requirements during this operation, as well as the care of the patient on the operating table. A haphazard performance of this type of operation, without the services of a trained team, must be condemned.

2. The operation is never undertaken without a full investigation of the patient's cardiac condition, which is examined by X-ray and electrocardiography, the state of his lungs (also confirmed by X-ray), and his renal and hepatic function. For the liver I am satisfied with a protein investigation, the albumin-globulin ratio, and the prothrombin index (the PI is becoming more essential than ever before in surgical procedures, because of the large numbers of people who are taking anti-coagulants, such as dicumarol, as well as its value as a test of liver function). For renal function I am satisfied with a blood urea and a chemical and microscopic examination of the urine. Naturally, a full blood count is done on every patient.

3. Every patient is hospitalized for 48 hours before operation, except in case of emergency. During these 48 hours his stomach is washed out twice daily with a saline solution until the washings are clear. During this pre-operative period the patient is also given penicillin and streptomycin twice daily and, if the prothrombin index is low, 3 doses of vitamin K at intervals. My theatre sister instructs the patient in this pre-operative period in his breathing exercises and limb exercises. He is instructed how to get in and out of bed with an abdominal wound, and she explains to him in great detail the nursing procedure which he has to undergo, viz. that the naso-gastric tube will be put down and left down for 12 hours before operation, that specimens of blood will be taken for tests, and that his blood will be grouped for blood transfusion. She also instructs him about his feeding—that he will have nothing by mouth for 24 hours after operation—and he is told how to wash his mouth out without swallowing. He is also taught how to turn from side to side, and how to maintain himself in a semi-Fowler position and assist the nursing staff in washing him and changing him. It is explained to him that he will be fed intravenously for 48 hours after the operation, and thereafter will receive a gradually increasing diet until the date of his discharge.

The night before the operation he is visited by my partner or myself, his confidence is established, and his fears allayed. A barbiturate sedative is given the night before operation.

4. *Post-operative care.* During the operation the patient receives 500 c.c. of blood, assuming that the blood count is normal and that the operation is not attended with any undue amount of shock or blood loss. The operation should not last more than 1-1½ hours; I am of the opinion that surgery beyond 1 hour requires major supplementary shock treatment and, as most of these operations are carried out on middle-aged persons, the risk increases with the duration of operation. Added to this, the efficiency of the surgery decreases rapidly after 1 hour.

When the patient returns from the theatre his bed is blocked at the foot, and as soon as he has recovered from the anaesthetic he is allowed sufficient pillows to bring him into a semi-Fowler position. It is my practice to use a draining jejunostomy in place of naso-gastric suction after surgery, a practice in which I follow Dr. Arthur Allen, of Boston. The jejunostomy is immediately connected to a bottle and drains dependently.

During the first 24 hours after operation the blood pressure and pulse rate are taken every 15 minutes for the first 2 hours, and every ½ hour for the next 6 hours. It is then taken every hour for the ensuing 24 hours, and thereafter twice daily for 3 days.

The patient is allowed as much omnopon as is required to keep him free of pain, and his breathing and limb exercises are commenced as soon as he is fully cooperative. At the conclusion of the blood infusion he is given 2,000 c.c. of invert sugar in water during the summer months, and 1,500 c.c. during the winter months. All this fluid flows in within 12 hours, i.e. a total of 2,000-2,500 c.c. in the first 24 hours. The drip is then removed and the patient is allowed to rest.

After 24 hours he is allowed ½ oz. of sterile water by the mouth hourly for the next 12 hours, and then 1 oz. of sterile water hourly for the following 12 hours, bringing us to 48 hours from the time of operation, whereafter he is allowed fluids by mouth *ad lib.*

In the 2nd post-operative day the patient is given a mixture consisting of 3 g. of potassium chloride and 4·5 g. of sodium chloride in 1,000 c.c. of invert sugar and water, plus further invert sugar and water to maintain him in a positive balance in relation to his fluid loss through the bladder and the jejunostomy drainage and his insensible loss. Once again this fluid is introduced

during daylight hours, and the drip is removed in the evening so that the patient has an undisturbed night.

On the morning of the 3rd day the drip is re-inserted and the same formula administered, whilst at this stage the patient is taking fluids *ad lib.* by mouth.

On the morning of the 4th day an assessment is made of whether the patient is able to continue with oral feeding alone. This will depend on whether bowel sounds are heard (they are now usually evident), and whether his intake by mouth is greater than his loss through the jejunostomy. If this is so, then the jejunostomy tube is raised to the level of his stomach and, if there is no spill over after 6 hours, the jejunostomy is then clamped off and on the afternoon of the 4th day the patient is allowed jelly, ice cream, and fluids *ad lib.*

On the morning of the 5th day he commences on a Meulengracht diet in small quantities, being fed every 2½ hours, and as soon as he has passed flatus is given ½ oz. of liquid paraffin morning and evening. The bowels usually act on the 4th or 5th day, and the patient's progress thereafter is undisturbed, being maintained on a Meulengracht diet until the 7th day when he is allowed a grilled chop, and on the 8th day he is allowed fillet steak with vegetables, not purée.

On the morning of the 9th day his stitches and jejunostomy tube are removed, and leakage from the jejunostomy usually ceases by the morning of the 10th day, when the patient may be discharged from the nursing home.

From 24 hours after operation the patient is got out of bed in the early morning before the drip is inserted, and in the evening after the drip has been removed, and from the morning of the 3rd day is encouraged to walk round his room. From the 4th day onwards he walks up and down the corridor assisted, until, usually by the 6th day, he is able to walk alone. He is encouraged to walk to the toilet from the 4th day onwards.

During this post-operative period my theatre sister calls on him on several occasions, and confers with the sister-in-charge on the adequacy of the calorie intake in his diet and on any symptoms of over-feeding or dumping; and a strict control is kept so that he is not allowed an excessive carbohydrate intake, which is often the cause of dumping.

The patient's haemoglobin is checked on the 7th day and, if necessary, a further blood transfusion is administered on this day.

During the first 4 post-operative days the patient is given injections of penicillin and streptomycin twice daily and of vitamins A, B and C once daily. On the morning of the 5th day vitamins A, B and C are administered by mouth.

He is visited by my partner or myself at least twice daily, and a routine examination of his lungs, abdomen and lower limbs is also made twice daily. In the event of any clinical disturbance of fluid or electrolyte balance, his electrolytes are determined, daily if necessary, and corrected accordingly. A strict adherence to this routine of pre- and post-operative treatment is one of the prime factors in what, I am pleased to say, are my satisfactory results.

The same applies to the operation. Each detail of the operation has been elaborately gone into with a view to standardizing the procedure, saving time, and minimizing trauma to tissue and shock, as well as post-operative complications.

*The Operation**

The type of procedure that I have adopted as a routine is the Polya-Hofmeister retrocolic iso-peristaltic type of partial gastrectomy, wherein 7/8ths of the stomach is removed. We have in latter years, where indication has presented itself, attempted the Billroth procedure in a number of cases, but I am satisfied that the results of the Billroth procedure, although it is said to be a more physiological operation, are certainly no better than those of the Polya-Hofmeister, and perhaps not as good. I am convinced that in a duodenal ulceration the Billroth procedure has no place.

The operation is done under general anaesthesia through a mid-line incision. The skin towels are stitched on in order to produce haemostasis of the skin edges and to ensure that infection from the skin will not enter the wound during operation. After a general exploration of the abdominal cavity the lesion is inspected and, if operation is proceeded with, the gastro-colic omentum is divided between haemostats and ligated with 40 linen thread. The right gastro-epiploic artery is individually ligated and the peritoneum dissected off the back of the pylorus. The right gastric artery is isolated and ligated with 25 linen thread and 2 catgut plain. Stay sutures are placed on either side of the duodenum, and the De Petz clamp is placed across the duodenum, thus sewing in two layers of metal clips.

The duodenum is then divided with the diathermy and the stump of duodenum invaginated with waxed-silk interrupted sutures. The cut end of the stomach is covered with a gauze swab tied into position. The left gastro-epiploic artery is now ligated and cut, and 2 short gastric arteries are ligated for gastric ulcer (3 for duodenal ulcer).

A safety ligature of 25 linen thread is then tied round the origin of the left gastric artery, and this artery is tied again with a double ligature where it curves forward onto the lesser curvature of the stomach. Another De Petz clamp is placed across the stomach and 3/4ths of the stomach removed for gastric ulcer (7/8ths for duodenal ulcer).

An opening is then made in the transverse meso-colon and the first portion of the jejunum is brought through this opening. The jejunum is stitched to the cut end of the stomach, the duodeno-jejunal flexure being approximated to the lesser curve, thus allowing of no afferent loop of jejunum. An anastomosis is performed leaving a large valve and a small anastomosis of not more than 1¼ inches. The whole anastomosis is then brought through into the greater sac and the opening in the meso-colon stitched round the stomach. A 22 catheter is next introduced into the jejunum 6 inches distal to the anastomosis, the catheter entering into the stomach, and this is brought out through a separate stab incision in the left loin through a hole in the omentum. The blood is then sucked out of the subdiaphragmatic spaces and the wound is closed in layers, the peritoneum with atraumatic chromic catgut no. 1 and the linea alba with interrupted 60 linen thread. The gastric suction tube is removed at the conclusion of the operation.

* This part of the address was illustrated with lantern slides.

CONCLUSION

I have attempted to outline what my opinion is about gastrectomy, and the procedure that I have adopted in my private practice. The same efficiency cannot be achieved in hospital practice because medical and nursing personnel have to be trained and routine cannot be standardized. There can be no unanimity amongst surgeons in this respect. I am satisfied that my results compare favourably with those recorded in the literature and, whilst not trying to impress upon you that this is the only procedure for gastrectomy, I have attempted to give you my views as far as I possibly can in the short time available. There are many aspects of this problem, particularly in regard to symptomatology, pathology, physiology, diagnosis, and so on, which for reasons of time I have not even attempted to touch on.

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4. Snyder, E. N. and Berne, C. J. (1957): Amer. J. Surg., **94**, 368.
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NATIONAL MEETING OF THE DERMATOLOGICAL SUB-GROUP (M.A.S.A.), JOHANNESBURG, 28 AND 29 MARCH 1959

NASIONALE VERGADERING VAN DIE DERMATOLOGIESE SUB-GROEP (M.V.S.A.), JOHANNESBURG, 28 EN 29 MAART 1959

President Dr. J. A. L. Leeming *President* *Guest-of-Honour* Dr. G. B. Dowling *Eregas*

The Dermatological Sub-group of the Medical Association of South Africa are holding a National Meeting in Johannesburg over the Easter week-end with Dr. G. B. Dowling, the eminent

Die Dermatologiese Sub-groep van die Mediese Vereniging van Suid-Afrika sal 'n Nasionale Vergadering te Johannesburg gedurende die Paasnaweek hou, met dr. G. B. Dowling, die bekende

British dermatologist, as guest-speaker. All practitioners are welcome to attend this meeting.

PROGRAMME : PROGRAM

Saturday 28 *March* 1959 *Maart* 28 *Saterdag*

Invitation Lectures : Lesings op Uitnodiging

Chairman—Dr. F. P. Scott—Voorsitter.

Venue : Vergaderplek: Board Room, Johannesburg General Hospital.
Raadsaal, Algemene Hospitaal, Johannesburg.

a.m. 10.30- 1.00 nm.: Dr. J. H. S. Gear—Virus infections of the skin.
Dr. F. Zumpt—The Tumbu fly, *Cordylobia anthropophaga* (Blanchard) in Southern Africa.
Dr. L. D. Erasmus—Scleroderma in gold miners.
Dr. H. D. Barnes—Porphyria in South Africa.

p.m. 1.00- 2.30 nm.: Luncheon—Noenmaal. Astor Hotel.

Clinical Demonstration : Kliniese Demonstrasie: p.m. 2.30-5.00 nm.
Chairman—Dr. G. H. Findlay—Voorsitter.

Venue : Vergaderplek: The General Section Hut, Johannesburg General Hospital : Hut vir Algemene Afdeling, Algemene Hospitaal, Johannesburg.

Banquet : Banket p.m. 7.30 nm. for/vir p.m. 8.00 nm. At the Langham Hotel, Johannesburg. All dermatologists and their wives or guests are welcome. Dr. J. G. Cowley will propose the toast of the guest-of-honour. Dr. Dowling will reply.

By die Hotel Langham, Johannesburg. Alle dermatoloë en hulle vrouens of gaste is welkom. Dr. J. G. Cowley sal die heildronk instel op die eregas. Dr. Dowling sal antwoord.

Britse dermatoloog, as gas-spreker. Alle geneeshere word by die vergadering verwelkom.

Sunday 29 *March* 1959 *Maart* 29 *Sondag*

Lectures by Dermatologists : Lesings deur Dermatoloë

Chairman—Dr. I. Gluckman—Voorsitter.

Venue : Vergaderplek: Board Room, Johannesburg General Hospital.
Raadsaal, Algemene Hospitaal, Johannesburg.

a.m. 10.00-12.30 nm.: Dr. G. B. Dowling—Defence mechanisms of the skin against alkaline substances.
Dr. J. J. Walker—Basalcell carcinoma. Speculations on possible factors influencing its low incidence amongst the non-European races of South Africa.
Dr. C. M. Ross—Poison ivy dermatitis.

If time and facilities are available, visiting members may care to project interesting slides at this point.
As tyd en omstandighede dit toelaat sal besoekende lede miskien ligskyfies op hierdie stadium kan wys.

Closing Luncheon : Noenmaal aan Einde: p.m. 1.00 nm.

Chairman—Dr. J. J. Jacobson—Voorsitter.

Venue : Vergaderplek: The Automobile Club, Killarney, Johannesburg.
Die Outomobiel-Klub, Killarney, Johannesburg.

All dermatologists and their wives or guests are welcome.
Alle dermatoloë en hulle vrouens of gaste is welkom.

For further information concerning arrangements for this meeting, please contact the Hon. Secretary, Dr. C. M. Ross, 309 Medical Centre, Pretorius Street, Pretoria.

Verdere inligting aangaande reëlings vir hierdie vergadering kan verkry word van die Ere-Sekretaris, dr. C. M. Ross, Mediese Sentrum 309, Pretoriusstraat, Pretoria.

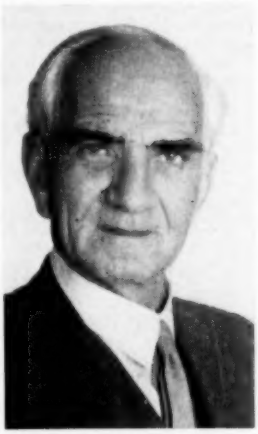
IN MEMORIAM

ARTHUR GEORGE HERBERT HAY-MICHEL, M.R.C.S., L.R.C.P.

Dr. R. J. W. Burrell, of East London, writes: 'Papa' Michel passed away on 23 January 1959 at the age of 85 years. Born in India, he spent his boyhood in Canada. A fervent ambition to become a doctor took him to London, but his studies were temporarily interrupted by the Anglo-Boer War, in which he saw active service as a trooper. Thereafter, he returned to London and whilst employed as an 'electrical assistant' at the Charing Cross Hospital he passed the Conjoint Examinations of the Royal Colleges of Surgeons and Physicians in April 1902.

His early professional life was spent in various parts of the world but the call to South Africa was irresistible. He practised at Theunissen, O.F.S., until the outbreak of the First World War, in which he served as a Captain, S.A.M.C., both in South-West and in East Africa. In 1919 he went to Cathcart, Cape, where he practised until a few years ago and then retired.

So dearly was Dr. Hay-Michel loved that residents throughout the Border called him 'Papa'. They will long remember him as a conscientious family doctor, a man of catholic interests and one of untiring energy for the common weal. It shall always be said of Dr. Hay-Michel that he was our friend.



Dr. Hay-Michel

Dr. R. Schaffer, of Queenstown, writes: The late Dr. Hay-Michel was an outstanding personality. He possessed a wide general knowledge and had wide cultural interests and human sympathies. In his younger days he was a competent surgeon who had a detailed knowledge of anatomy. He was an expert artist and made many excellent anatomical sketches of pathological and anatomical abnormalities encountered in his surgical practice.

He was a general practitioner of the old school—kind, courteous and understanding. Many patients had reason to be grateful for his expert advice. He was the beloved family doctor.

Hay-Michel was a founder member of the Medical Association of South Africa and he held high office both in the Queenstown Division and the Border Branch. Until age and indisposition made travel difficult he was a regular attendant at Branch meetings, at Division meetings and at meetings of the Branch Council. On many occasions he travelled to Queenstown, to East London and to other centres in the interests of the Association. He organized several clinical meetings at the Cathcart Hospital, which were always most interesting and well attended.

Born in Delhi, India, on 7 May 1876, he spent his boyhood in Canada. He went to London to study medicine but interrupted his studies during the Boer War, in which he saw active service as a trooper. After demobilization he returned to London, and while employed at Charing Cross Hospital he passed the Conjoint Examinations of the Royal College of Physicians and Surgeons in 1902.

After practising in various parts of the world he came to South Africa and opened a practice at Theunissen, O.F.S. There he

practised until the outbreak of World War I, when he joined the S.A.M.C., serving with the rank of Captain in South West Africa and later in East Africa. His friends, Dr. W. Paisley of Queenstown and Dr. A. W. Burton of King Williams Town, served with him in this campaign and can tell many interesting anecdotes of this period.

In 1919 he went to Cathcart and he practised there until his death on 23 January 1959 at the age of 85 years.

His was an interesting and eventful life. He served his patients, his Association and the community in which he lived. He was loved and respected and will long be remembered. Sincere sympathy is expressed to his widow.

Dr. G. H. Alabaster, formerly of the Union-Castle Line, writes: May I be allowed to add my tribute to the well-loved memory of a fine man and a doctor whose capability and character were alike outstanding. His goodness and kindness I experienced through a period of 37 years.

Dr. Hay-Michel, who came of Channel Islands stock, was a member of a large family. His father had distinguished himself by service to the Indian Government, and his earlier years were spent in India. His later school education was completed at Rugby, from where he went to Germany to commence his University education at Heidelberg, in the Faculty of Engineering.

From Heidelberg he returned to London, having transferred his allegiance to medicine, and entered as a student at King's College Hospital, London. There he enlisted for service in the South African War, where on one occasion when out on patrol, he was narrowly missed by a bullet which killed the man riding beside him.

After qualifying in London Dr. Hay-Michel held appointments in the Northern Transvaal and in the Orange Free State. When acting as District Surgeon at Winburg, O.F.S., he was instrumental in collecting some hundreds of rifles at the time of the rebellion, and was personally thanked by General Botha for his services. He then took part in the South West African and East African campaigns of the World War I. During the war he contracted a severe post-malarial illness, as a result of which he settled at Cathcart, in the Cape. He played a leading part in establishing a hospital there, and during his early years of practice was conspicuous in his efforts to bring about the foundation of the Medical Association of South Africa.

His clinical work was habitually careful and thoughtful. A unique tinted drawing which he made of a case of bowel strangulation through a ring made by the adhesion of the tip of the appendix to the caecum, was an example of his care in detail. This drawing won the distinction of notice outside South African territory and eventually was sent, on request, to a Russian professor of surgery.

Dr. Hay-Michel was not only an outstandingly good doctor, but was also a kind and generous man to whom money meant little in comparison to work and friendship. It was one of his sayings that God should be spelt with two 'O's. He will be long remembered for the quality both of his services and of his friendship.

The President (Dr. R. Schaffer) represented the Medical Association at the funeral, which Dr. W. Paisley, Dr. D. M. Holmes, Dr. A. W. Burton, Dr. J. R. Norton and Dr. R. J. W. Burrell also attended. Dr. J. H. Swift was one of the pallbearers.

PASSING EVENTS : IN DIE VERBYGAAN

Dr. C. W. Louw, M.B., Ch.B., voorheen van Alexandria, Kaap, praktiseer nou te Pictonstraat 6, Parow, Kaap.

* * *

Dr. I. Orr, M.B., B.Ch., Dip. O. & G. (Rand), of Johannesburg, has recently returned from a visit to London, for postgraduate study, where he was awarded the M.R.C.O.G. Dr. Orr has now resumed his practice as a specialist obstetrician and gynaecologist at 506 Medical Arts Building, Jeppe Street, Johannesburg. Telephones: Rooms 22-0035, residence 44-5001.

* * *

The Sixth International Congress for Internal Medicine will be held in Basel, Switzerland, from 24-27 August 1960. This Congress will be organized in conjunction with the Swiss Society for Interna Medicine. For further details apply to the Secretariat of the 6th International Congress for Internal Medicine, 13 Steinentorstrasse, Basel, Switzerland.

* * *

Correction. In the article *A Programme for the Care of Cripples in South Africa* by Mr. G. T. du Toit, which appeared on page 163 of the *Journal* of 21 February 1959, a correction is to be made on line 21 on page 164. This should read as follows: ' . . . that the country is losing at least £100,000,000 of national wealth per year'.

NEW PREPARATIONS AND APPLIANCES : NUWE PREPARATE EN TOESTELLE

EMBELIX

Maybaker (S.A.) (Pty.) Ltd. announce that Embelix brand general tonic is now available with an improved formulation and a new flavour of appeal to children as well as adults, and they supply the following information: The formula includes caffeine citrate B.P.C. 0·21 g., potassium glycerophosphate solution B.P.C. 1·84 g., sodium glycerophosphate solution 0·92 g., aneurine hydrochloride B.P. 0·018 g., nicotinic acid B.P. 0·088 g., and riboflavine-5-phosphate sodium 0·024 g.

Embelix is of value in convalescence when a good general tonic is needed to hasten the patient's return to full health. It is particularly useful in patients recuperating from long illness involving the digestive system. 'Embelix' also helps to build up the well-being of patients whose illness may have left them with a feeling of lethargy.

Embelix is supplied in bottles of 8 and 25 fl. oz.

ALUDROX SA TABLETS

Since the discovery of the therapeutic properties of aluminium hydroxide gel, Wyeth Laboratories have been a pioneer in the development of medicaments for peptic ulcer. Now, Wyeth research presents Aludrox SA.

Aludrox SA tablets (aluminium hydroxide with magnesium hydroxide, ambutonium bromide, and butabarbital) benefit the peptic-ulcer patient by providing complete medical management. It relieves the pain, reduces the acid secretion, calms the emotional distress, and promotes ulcer healing.

Recent studies conducted by 333 physicians and involving 638 patients with ulcers and other gastric conditions revealed that 85·7% of those treated with Aludrox SA experienced 'goodl to excellent' relief. Another 7·2% received some relief. A later report concerning 343 members of the group tested stated that the conditions of the patients ranged from 'healed' to 'symptoms improved'.

Aludrox SA combines antacid, sedative, and anticholinergic action to meet complete medical management in a single preparation dosage form. Aludrox SA tablets are available on prescription only, packed in bottles of 60 tablets.

ANCOFEN

British Drug Houses announce the addition of Ancofen to their range of products, and supply the following information: Ancofen is regarded as an advance in the treatment of migraine and other forms of headache of a paroxysmal nature. By the use of Ancofen the 4 principle groups of symptoms associated with migraine, i.e. headache, sensory symptoms which are usually visual in nature, nausea, and vomiting accompanied by malaise and lassitude, are effectively controlled.

Ancofen has a three-fold action. Meclozine hydrochloride 10 mg. exerts a sedative action on the nervous system, acts as an

anti-emetic and eliminates any element of hypersensitivity. Ergotamine tartrate 1 mg. increases the tone of blood-vessel walls and decreases the peculiar throbbing quality of the migraine headache. Caffeine 100 mg. is included as the third constituent of Ancofen since it potentiates the efficacy of ergotamine.

The usual dose in the prodromal stage is 1 tablet hourly to a total of 6 tablets in order to halt the attack. Should an attack develop before Ancofen can be taken, a dose of 2 tablets hourly to a total of 6 tablets is prescribed.

A further advantage of Ancofen is that it is well tolerated by the patient and is generally free from side-reactions. The tablets are conveniently packed in tubes of 10 or bottles of 50 tablets.

BOOKS RECEIVED: BOEKE ONTVANG

The Care of the Geriatric Patient. Edited by E. V. Cowdry, Ph.D., Sc.D. (Hon.). Pp. 438. South African Price 68s. St. Louis: The C. V. Mosby Company. 1958.

Injuries and Surgical Diseases of the Ischium. By Henry Milch, M.D. Pp. x+163. 106 Figures. $10.50. New York: Paul B. Hoeber, Inc. 1958.

Clinical Endocrinology. 2nd Edition. By Karl E. Paschkis, M.D., Abraham E. Rakoff, M.D. and Abraham Cantarow, M.D. Pp. xii+941. 274 Illustrations, 6 in full colour. $18.00. New York: Paul B. Hoeber Inc. 1958.

Advances in Pediatrics. Volume X. Editor: S. Z. Levine. Pp. 362. Illustrations. $9.00. Chicago: Year Book Publishers, Inc. 1958.

Recent Advances in Paediatrics. 2nd Edition. Edited by Douglas Gairdner, D.M., F.R.C.P. Pp. viii+378. 82 Illustrations. 48s. net. London: J. & A. Churchill Ltd. 1958.

Recent Advances in Cerebral Palsy. Edited by R. S. Illingworth, M.D., F.R.C.P., D.P.H., D.C.H. Pp. x+389. 136 Illustrations. 50s. net. London: J. & A. Churchill Ltd. 1958.

The Management of Emergencies in Thoracic Surgery. By John Borrie, M.B.E., Ch.M., F.R.C.S. (Eng.), F.R.A.C.S. Pp. xi+340. Illustrations. New York: Appleton-Century-Crofts, Inc. 1958.

Pathophysiology in Surgery. By James D. Hardy, M.S., M.D., F.A.C.S. Pp. xx+704. 278 Figures. $19.00. Baltimore: The Williams & Wilkins Company. 1958.

The Devil's Repertoire or Nuclear Bombing and the Life of Man. By Victor Gollancz. Pp. 192. 10s. 6d. Bound in Cloth and 5s. in Paper Covers. London: Victor Gollancz Ltd. 1958.

Insect Resistance and Vector Control. Eighth Report of the Expert Committee on Insecticides. Technical Report Series No. 153. Pp. 67. 3 Figures. 3s. 6d. Also published in French and Spanish. Geneva: World Health Organization. 1958. Local Sales Agent: Van Schaik's Bookstore (Pty.) Ltd., P.O. Box 724, Pretoria.

Joint FAO/WHO Expert Committee on Nutrition. Fifth Report. Technical Report Series No. 149. Pp. 55. 3s. 6d. Also available in French and Spanish. Geneva: World Health Organization. 1958. Local Sales Agent: Van Schaik's Bookstore (Pty.) Ltd., P.O. Box 724, Pretoria.

The Chemical Prevention of Cardiac Necroses. By Hans Selye, M.D., Ph.D., D.Sc. Pp. ix+235. 20 Figures. $7.50. New York: The Ronald Press Company. 1958.

Obstetrical Practice. 7th Edition. By Alfred C. Beck, M.D. and Alexander H. Rosenthal, M.D. Pp. xiii+1115. Illustrated. 112s. London: Baillière, Tindall and Cox Ltd. 1958.

A Symposium on the Evaluation of Drug Toxicity. Edited by A. L. Walpole, Ph.D., B.Sc. and A. Spinks, M.A., Ph.D., B.Sc. Pp. xi+138. 58 Illustrations. 25s. net. London: J. & A. Churchill Ltd. 1958.

Biochemistry and the Central Nervous System. 2nd Edition. By Henry McIlwain, Ph.D., D.Sc. Pp. vii+288. 45 Illustrations. 45s. net. London: J. & A. Churchill Ltd. 1959.

The Year Book of Pediatrics 1958-59. Edited by Sydney S. Gellis, M.D. Pp. 496. 125 Figures. $7.50. Chicago: Year Book Publishers, Inc. 1958.

Staphylococcal Infections. By Ian Maclean Smith, M.D., Ch.B., F.R.F.P.S.G. Pp. 180. 6 Figures. $4.25. Chicago: Year Book Publishers, Inc. 1958.

Urology in Outline. By T. L. Chapman, Ch.M., F.R.C.S. (Eng.), F.R.F.P.S. (Glas.). Pp. vii+176. 138 Figures. 27s. 6d. + + 1s. 7d. Postage Abroad. Edinburgh and London: E. & S. Livingstone Ltd. 1959.

Personality Change and Development as Measured by the Projective Techniques. By Molly Harrower, Ph.D. Pp. 383. Illustrations. $10.00. New York and London: Grune & Stratton, Inc. 1958.

Problems of Addiction and Habituation. Edited by Paul H. Hoch, M.D. and Joseph Zubin, Ph.D. Pp. xii+250. Illustrations. $6.50. New York and London: Grune & Stratton, Inc. 1958.

Physiology of Spinal Anesthesia. By N. M. Greene, B.S., M.A., M.D. Pp. xi+195. Illustrations. $6.00. Baltimore: The Williams & Wilkins Company. 1958.

Recent Studies in Epidemiology. Edited by J. Pemberton, M.D., M.R.C.P., D.P.H. and H. Willard, M.D. Pp. xiii+203. Illustrations. 25s. net. Oxford: Blackwell Scientific Publications. 1958.

Lipidoses. Diseases of the Intracellular Lipid Metabolism. 3rd Edition, Revised and Enlarged. By S. J. Thannhauser, M.D., Ph.D., Hon. M.D. Pp. xiv+600. 126 Figures. $19.75. New York and London: Grune & Stratton, Inc. 1958.

Treatment of Cancer and Allied Diseases. 2nd Edition. Volume I. *Principles of Treatment.* By 55 Authors. Edited by George T. Pack, M.D., F.A.C.S. and Irving M. Ariel, M.D., F.A.C.S. Pp. xxi+646. 505 Illustrations. $22.50. New York: Paul B. Hoeber, Inc. 1958.

Glasgow's X-ray Campaign against Tuberculosis, 11 *March*-12 *April* 1957. Pp. 117. Illustrations. Edinburgh: Department of Health for Scotland. 1958.

Hématologie Clinique. Par Jean Bernard et Marcel Bessis. Pp. 526. 295 Figures. 37 Planches hors texte en couleurs (20×26). 14.50 fr. Paris: Masson et Cie. 1958.

Staphylococcus Pyogenes and its Relation to Disease. By Stephen D. Elek, M.D., D.Sc., Ph.D., D.P.H. Pp. vii+767. 44 Figures. XXII Tables. 84s. net + 3s. Postage Abroad. Edinburgh and London: E. & S. Livingstone Ltd. 1959.

Medical History of the Second World War. The Royal Air Force Medical Services. Volume III. *Campaigns.* Edited by S. C. Rexford-Welch, M.A., M.R.C.S., L.R.C.P., R.A.F. Pp. xxv+730. LXVII Plates. 57 Maps. 37 Figures. 105s. net. London: Her Majesty's Stationery Office. 1958.

Cyclopropane Anesthesia. 2nd Edition. By Benjamin Howard Robbins, B.A., M.S., M.D. Pp. xii+293. 74 Figures. $9.00. Baltimore: The Williams and Wilkins Company. 1958.

Haematological Technique. 2nd Edition. By E. M. Darmady, M.A., M.D. (Camb.), F.R.C.P. and S. G. T. Davenport, F.I.M.L.T. Pp. viii+244. 4 Coloured Plates and 23 Text Figures. 24s. net. London: J. & A. Churchill Ltd. 1958.

Neoplastic Disease at Various Sites. General Editor: D. W. Smithers, M.D., F.R.C.P., F.F.R. Volume II. *Tumours of the Bladder.* Edited by David M. Wallace, O.B.E., M.S., F.R.C.S. Pp. xvi+352. 202 Figures. 60s. net + 2s. 8d. Postage Abroad. Edinburgh and London: E. & S. Livingstone Ltd. 1959.

The ABO Blood Groups. Comprehensive Tables and Maps of World Distribution. By A. E. Mourant, M.A., D.Phil., D.M. (Oxford), M.R.C.P. (London), Ada C. Kopeć, D. ès Sc. (Genève) and Kazimiera Domaniewska-Sobczak, M.A. (Wilno), A.L.A. Pp. viii+276. 6 Maps. 42s. net. Oxford: Blackwell Scientific Publications. 1958.

Practical Blood Grouping. By F. Stratton, M.D., D.Sc., D.P.H. and P. H. Renton, M.D., B.Sc. Pp. xxiv+331. Illustrations. 42s. Oxford: Blackwell Scientific Publications. 1958.

Bailey's Text-book of Histology. 14th Edition. Revised by Wilfred M. Copenhaver, Ph.D. (Editor) and Dorothy D. Johnson, Ph.D. Pp. xiii+633. 478 Illustrations. 88s. net. London: Baillière, Tindall and Cox Ltd. 1958.

A Synopsis of Hygiene (Jameson and Parkinson). 11th Edition. By Llywelyn Roberts, M.D., M.R.C.P., D.P.H., assisted by Kathleen M. Shaw, M.B.E. Pp. viii+694. 22 Illustrations. 60s. net. London: J. & A. Churchill Ltd. 1958.

Text-book of Physiology and Biochemistry. 4th Edition. E. George H. Bell, B.Sc., M.D. (Glasg.), F.R.F.P.S.G., F.R.S.E.,

J. Norman Davidson, M.D., D.Sc. (Edin.), F.R.F.P.S.G., F.R.I.C., F.R.S.E. and Harold Scarborough, M.B., Ph.D. (Edin.), F.R.C.P.E., M.R.C.P.E. Pp. xi+1065. Illustrations. 63s. net + 4s. 2d. Postage Abroad. Edinburgh and London: E. & S. Livingstone Ltd. 1959.

Anatomy for Surgeons. Vol. 3. *The Back and Limbs.* By W. Henry Hollinshead, Ph.D. Pp. xii+901. 785 Illustrations. $23.50. New York: Paul B. Hoeber, Inc. 1958.

Outline of Fractures including Joint Injuries. 2nd Edition. By John Crawford Adams, M.D., F.R.C.S. Pp. viii+268. 237 Figures. 27s. 6d.+1s. 7d. Postage Abroad. Edinburgh and London: E. & S. Livingstone Ltd. 1958.

Surgery of the Head and Neck. A Handbook of Operative Surgery. By Robert A. Wise, M.D. and Harvey W. Baker, M.D. Pp. 319. 82 Plates. $9.75. Chicago: Year Book Publishers, Inc. 1958.

Clinical Obstetrics and Gynecology. Vol. I, No. 3. September 1958. *Symposium on Special Diagnostic Aids.* Edited by C. Paul Hodgkinson, M.D. *Symposium on Abnormal Uterine Bleeding.* Edited by John I. Brewer, M.D. This is the third number of a quarterly series. Pp. 304. Illustrations. $18.00 for 4 consecutive numbers. New York: P. B. Hoeber, Inc. 1958.

The Year Book of Obstetrics and Gynecology 1958-59. Edited by J. P. Greenhill, B.S., M.D., F.A.C.S., F.I.C.S. Pp. 607. 50 Figures. $7.50. Chicago: Year Book Publishers, Inc. 1958.

A Practice of Thoracic Surgery. 2nd Edition. By A. L. d'Abreu, O.B.E., Ch.M., F.R.C.S. Pp. viii+619. Illustrations. 100s. net. London: Edward Arnold (Publishers) Ltd. 1958.

The Practice of Sanitation. 3rd Edition. By Edward Scott Hopkins and Wilmer Henry Schulze. Pp. ix+487. 141 Figures. $8.00. Baltimore: The Williams & Wilkins Company. 1958.

Neo-natal Paediatrics. Edited by W. R. F. Collis, M.D., F.R.C.P., F.R.C.P.I., D.P.H. Pp. xiii+301. 35 Figures. 30s. net. London: William Heinemann—Medical Books—Ltd. 1958.

An Atlas of Surgery. By F. Wilson Harlow, M.B., B.S. (Durham), F.R.C.S. (Eng.). Pp. xiii+363. 1,292 Illustrations. 50s. net. London: William Heinemann—Medical Books—Ltd. 1958.

British Obstetric and Gynaecological Practice. 2nd Edition. Edited by Sir Eardley Holland, M.D. (Lond.), F.R.C.P., F.R.C.S., F.R.C.O.G. and Aleck Bourne, M.A., M.B., B.Ch. (Cantab.), F.R.C.S., F.R.C.O.G. Pp. xii+891. Illustrations. 105s. net. London: William Heinemann—Medical Books—Ltd. 1958.

Symposium on Nuclear Sex. Edited by the Organizing Committee by the Secretary, D. Robertson Smith, M.A., M.D. and William M. Davidson, M.D. Pp. xvii+188. 14 Figures. 21s. net. London: William Heinemann—Medical Books—Ltd. 1958.

Diagnostic Anatomy. By Weston D. Gardner, M.D. Pp. 376. 20 Figures. 85s. St. Louis: The C. V. Mosby Company. 1958.

Etiology and Treatment of Leukemia. Proceedings of the First Louisiana Cancer Conference. Edited by Walter J. Burdette, Ph.D., M.D., F.A.C.S. Pp. 167. 14 Figures. South African Price 34s. St. Louis: The C. V. Mosby Company. 1958.

Rehabilitation Medicine. A Text-book on Physical Medicine and Rehabilitation. By Howard A. Rusk, M.D. and 36 collaborators. Pp. 572. 172 Illustrations. 102s. St. Louis: The C. V. Mosby Company. 1958.

A Method of Anatomy—Descriptive and Deductive. 6th Edition. By J. C. Boileau, M.C., M.B., Ch.B., F.R.C.S. (Edin.). Pp. vii+879. Illustrated. 88s. London: Baillière, Tindall and Cox Ltd. 1958.

Surgery of the Sympathetic Nervous System. 3rd Edition. By Sir James Paterson Ross, K.C.V.O., LL.D., M.S., F.R.C.S., F.R.A.C.S., F.A.C.S. Pp. xii+170. Illustrated. 35s. net. London: Baillière, Tindall and Cox Ltd. 1958.

Text-book of Surgery. 2nd Edition. By Patrick Kiely, B.Sc., M.D., M.Ch. (N.U.I.), F.R.C.S. (Eng.). Pp. x+1,158. 605 Illustrations. 63s. net. London: H. K. Lewis & Co. Ltd. 1958.

Heredity of the Blood Groups. By Alexander S. Wiener, A.B., M.D., F.A.C.P., F.C.A.P. and Irving B. Wexler, A.B., M.D., F.A.C.P. Pp. x+150. 4 Figures. $6.00. New York and London: Grune & Stratton, Inc. 1958.

Die Blutgruppen des Menschen. (Blood Groups in Man, 3rd Edition). Von R. R. Race, Ph.D. und R. Sanger, Ph.D., B.Sc. Deutsche Übersetzung von Prof. Dr. O. Prokop. Pp. xx+372. 31 Abbildungen. 107 Tabellen. Ganzleinen DM 39.80. Stuttgart: Georg Thieme Verlag. 1958.

The Comparative Anatomy and Physiology of the Nose and Paranasal Sinuses. By Sir Victor Negus, Hon. D.Sc., M.S., F.R.C.S. (Eng.), Hon. F.R.C.S. (Edin.), Hon. F.R.C.S. (Ireland). Pp. xv+402. 178 Figures. 70s. net+3s. Postage Abroad. Edinburgh and London: E. & S. Livingstone Ltd. 1958.

Le Placenta Humain. Aspects Morphologiques et Fonctionnels. Par le Professeur Jean Snoek et collaborateurs. Pp. 718. 175 Figures. 48 Tableaux. 7.500 fr. Paris: Masson et Cie. 1958.

The Preservation of Eyesight. Prepared with the advice of a Consultative Committee and edited by Sir Arthur Salusbury MacNalty, K.C.B., M.A., M.D. (Oxon.), F.R.C.P. (Lond.), F.R.C.S. (Eng.), D.P.H., Hon. F.R.S. (Edin.). Pp. vii+107. 9 Illustrations. 12s. 6d.+9d. postage. Bristol: John Wright & Sons Ltd. 1958.

A Laboratory Manual of Abnormal Haemoglobins. Prepared under the direction of J. H. P. Jonxis and T. H. J. Huisman for the Council for International Organizations of Medical Sciences and the Middle East Science Cooperation Office of Unesco. Pp. 39. 12 Figures. 9s. 6d. Oxford: Blackwell Scientific Publications. 1958.

Lehrbuch der Kinderchirurgie. Von Priv.-Doz. Dr. M. Grob. xii+775 Seiten. 876 Zum Teil mehrfarbige Abbildungen in 1310 Einzeldarstellungen. Ganzleinen DM 157.00. Stuttgart: Georg Thieme Verlag. 1958.

Leistungen und Ergebnisse der neuzeitlichen Chirurgie. Von Emil K. Frey. xii+339 Seiten. 154 Abbildungen. 52 Tabellen. Ganzleinen DM 84.00. Stuttgart: Georg Thieme Verlag. 1958.

Die Pränatalen Infektionen des Menschen, unter besonderer Berücksichtigung von Pathogenese und Immunologie. Von Dr. H. Flamm. xii+136 Seiten. 2 Abbildungen. DM 19.80. Stuttgart: Georg Thieme Verlag. 1958.

Centaur: Essays on the History of Medical Ideas. By Félix Martí-Ibánez, M.D. Pp. xvii+716. 86.00. New York: MD Publications Inc. 1959.

Schmerzlose Geburt durch Psychoprophylaxe. Von Dr. F. Roth. viii+124 Seiten. 8 Abbildungen. DM 12.00. Stuttgart: Georg Thieme Verlag. 1958.

Grosse Nervenärzte. Band II. 22 Lebensbilder. Herausgegeben von Kurt Kolle. x+252 Seiten. 21 Abbildungen. Ganzleinen DM 29.40. Stuttgart: Georg Thieme Verlag. 1959.

Die medikamentöse Behandlung der Schilddrüsenerkrankungen. Von Prof. Dr. W. Grab und Prof. Dr. K. Oberdisse. xii+285 Seiten. 55 Abbildungen. 14 Tabellen. DM 39.60. Stuttgart: Georg Thieme Verlag. 1959.

Postmortale klinisch-chemische Diagnostik und Todeszeitbestimmung mit chemischen und physikalischen Methoden. Von Prof. F. Schleyer. viii+64 Seiten. 34 Abbildungen. DM 14.70. Stuttgart: Georg Thieme Verlag. 1958.

Leitfaden der Blutmorphologie—Manual of Blood Morphology. Von L. Scudel. Englische und französische Übersetzung von J. Meng. 9 Verbesserte Auflage. 54 Seiten. 18 farbige Tafeln. DM 13.30. Stuttgart: Georg Thieme Verlag. 1958.

Physiologie des Bewusstseins in entwicklungsgeschichtlicher Betrachtung. Von Prof. Dr. U. Ebbecke. xii+211 Seiten. Ganzleinen DM 27.00. Stuttgart: Georg Thieme Verlag. 1959.

Akute innere Krankheiten. 2 Erweiterte und verbesserte Auflage. Diagnostische und therapeutische Hinweise in tabellarischer Übersicht. Von Prof. Dr. H. A. Kühn, Doz. Dr. H. Klepsig und Dr. E. Schildge. viii+247 Seiten. DM 16.50. Stuttgart: Georg Thieme Verlag. 1959.

Lehrbuch der Auskultation und Perkussion. 2 Auflage. Von Prof. Dr. K. Holldack. xii+196 Seiten. 85 Abbildungen. DM 16.50. Stuttgart: Georg Thieme Verlag. 1959.

Ergebnisse der gesamten Tuberkulose- und Lungenforschung. Band XIV. Herausgegeben von St. Engel, L. Heilmeyer, J. Hein und E. Uehlinger. viii+736 Seiten. 283 Abbildungen in 462 Einzeldarstellungen. Ganzleinen DM 144.00. Stuttgart: Georg Thieme Verlag. 1958.

Atlas of Intracardiac Pressure Curves. By Otto Bayer and Hans Helmut Wolter. Pp. xvi+185. 55 Figures. 42 Tables. DM 68.00. Stuttgart: Georg Thieme Verlag. 1959.

BOOK REVIEWS : BOEKBESPREKINGS

INFERTILITY

Human Infertility. By C. Lee Buxton, M.D., Med. Sc.D. and Anna L. Southam, M.D. With a Chapter on Endometrial Diagnosis by Earl T. Engle, Ph.D. Pp. x+229. 43 Figures. $7.50. New York: Paul B. Hoeber, Inc. 1958.

Human infertility is still one of the most complex problems the profession has to deal with. This publication covers the subject very widely and thoroughly. The authors' arguments are all very sound and important, simple and basic. Their approach, above all, is honest and they do not profess to have found any new therapy for the condition.

The chapter on endometrial interpretation by such a renowned worker as the late E. T. Engle, must add prominence to this work. The clinical material presented in the publication is based on patients seen at the Sloane Hospital Infertility Clinic and in the authors' private practice. The total number of cases analysed was 2,053 but those followed-up for a year or more, totalled 1,568. The figures submitted are well presented and of great value to other workers in this field.

The importance of the infertile couple to be treated as a unit is very strongly emphasized. This procedure has been accepted in the USA and is borne out by the fact that the Specialty Board allows the gynaecologist to accept and treat the infertile couple as a unit.

Except for the usual 'Americanisms', the book is extremely well written and provides easy and enjoyable reading. It is printed on high-gloss paper and the illustrations are excellent.

J.A.

VERLOSKUNDIGE PRAKTYK

Obstetrical Practice. 7e druk. Deur Alfred C. Beck, M.D. en Alexander H. Rosenthal, M.D. Pp. xiii+1115. Illustrasies. $14.00. Baltimore: The Williams & Wilkins Company. 1958

Die sewende uitgawe van hierdie Amerikaanse teksboek en naslaanboek in verloskunde het in 1958 verskyn. Hierdie boek vergelyk baie goed met ander standaardwerke. Die teks lees maklik want alle stellinge word breedvoerig verduidelik. Illustrasies is goed en volop. Aan die einde van elke hoofstuk is daar 'n volledige bibliografie. Die fisiologiese aspekte van swangerskap en kraam word genoegsaam beklemtoon.

Om tred te hou met die nuutste navorsingsresultate en ontwikkelings in obstetriese praktyk is daar 'n nuwe hoofstuk geskrywe oor plasentale fisiologie. Daar word verder gewaarsku teen onnodige roetine X-straalondersoeke in swangerskap.

Interessante nuwerwetse idees, wat wel opgeneem is in hierdie boek, is o.a. roetine binneaarse voeding in die eerste en tweede stadia van kraam, en die gebruik van binneaarse pitocin vir uterine disfunksie in die eerste stadium.

In die behandeling van die derde stadium word die Brandt-Andrews-metode van die plasenta uitdruk aangeraai, terwyl Crede se metode glad nie eers genoem word nie.

Daar is verder ook nuwe hoofstukke oor prolaps van die naelstring, veelvoudige swangerskap, mola hidatosa, hidramnios en perinatale mortaliteit.

Die persoon wat kan bekostig om hierdie boek te koop, kry waarde vir sy geld. Dit kan aanbeveel word by voorgraadse en nagraadse studente, sowel as by algemene praktisyns en spesialiste in die verloskunde.

J.N. de V.

HISTOPATHOLOGICAL TECHNIQUE

Handbook of Histopathological Technique. (Including Museum Technique.) By C. F. A. Culling, F.I.M.L.T., F.R.M.S. Pp. x+446+(27). 79 Figures. 51s. 9d.+1s. 8d. Postage. London: Butterworth & Co. (Publishers) Ltd. South African Office: Butterworth & Co. (Africa) Ltd., P.O. Box 792, Durban. 1957.

This book, written in a simple and basic style, is primarily intended for the training of medical technologists, and as such covers the field of histological practice very adequately. In addition, however, practising histologists, no matter what their professional status, will find it of considerable use. That the author is a man of considerable experience is immediately reflected in its contents. The descriptions of techniques and the discussion of practical difficulties that arise, and the means of overcoming them, can only come from an individual long familiar with his subject. It is probably this quality most of all that recommends the book.

The chapters that deal with section cutting and museum techniques are particularly useful. Very often, in other contemporary works, a discussion of section cutting is confined only to the simplest descriptions of apparatus and museum techniques are omitted. Section cutting, probably the most important single procedure, is given the attention it deserves.

Many technologists, who commence their training without the benefit of preliminary courses in anatomy and physiology, must encounter a bewildering flood of new terms and phrases. For them the introductory and explanatory notes which preface certain chapters must also serve a useful purpose.

The staining procedures described are standard ones, and despite the fact that histochemistry is really beyond the province of this book, it is remarkable how much of it is covered.

The sections on special procedures deal with autoradiography, vital staining, micro-incineration and injection techniques, but do little more than outline the principles involved in these procedures. Although a section is devoted to microscopy, these are really too skimpy to be of real use to anyone.

It is disappointing that a chapter on the principles of photography, with special reference to macrophotography and photomicrography, has not been included in a book otherwise as comprehensive as this. Other than its role in teaching, publication and research, photography, from the point of view of recording, forms an indispensable procedure to even the humblest routine histological laboratory, and no technologist can be regarded as adequately trained unless he has some knowledge of it.

The contents of this book is of such a fundamental nature that it could profitably be included in the library of every histological laboratory.

C.J.U.

CHEMOTHERAPY OF TUBERCULOSIS

The Chemistry and Chemotherapy of Tuberculosis. A compilation and critical review of existing knowledge on the chemistry of tubercle bacilli and their products, chemical changes and processes in the host, and chemical aspects of the treatment of tuberculosis. 3rd edition. By Esmond R. Long, M.D., Ph.D., Sc.D. Pp. xviii+450. Illustrations. Sterling price 96s. Published in England by Baillière, Tindall & Cox Ltd., London. $12.00. Baltimore: The Williams & Wilkins Company. 1958.

The 3rd edition of this review by Prof. E. R. Long represents the gleaned information, gathered over a period of 35 years, of our present knowledge of tuberculosis.

For those engaged in anti-tuberculosis work, this book will be invaluable, since it extracts the essentials from a voluminous literature and allows the reader to grasp the facts, while presenting him with lists of the original articles.

The general layout of the book is excellent and every chapter is provided with its own list of references.

As a critical review of our present knowledge of tuberculosis, its cause and effect, this book fulfils its purpose; and it is, moreover, a handy reference book on the subject as well, thanks to the excellent indexing.

J.B.P.

DISEASES OF WOMEN

Diseases of Women. 10th edition. By 10 teachers under the direction of F. W. Roques, C.B.E., M.D., M.Chir., F.R.C.S., F.R.C.O.G., edited by F. W. Roques, John Beattie and Joseph Wrigley. Pp. viii+556. 209 figures. 36s. net. London: Edward Arnold (Publishers) Ltd. 1959.

It is 40 years since this book was first published and it must have been found of great value to many thousands of students. Not only have there been 9 previous editions, but there have been 10 additional reprintings.

The present editors have maintained the effort of their predecessors to keep pace with developments in the practice of gynaecology, and while several chapters have been expanded, a section on vaginal cytology has been included.

This edition has some 75 more pages and 32 more figures than the last edition, and there is no doubt that it will meet with the same success as those it follows.

A.H.T.

THE MEDICAL ANNUAL

The Medical Annual. A Year Book of Treatment and Practitioners' Index. Editors: Sir Henry Tidy, K.B.E., M.A., M.D. (Oxon), F.R.C.P. and R. Milnes Walker, M.S. (Lond.), F.R.C.S. Pp. xl+580+23+4. XLII plates. 30 figures. 42s. + 1s. 9d. postage. Bristol: John Wright & Sons Ltd. 1958.

This Medical Annual appears under a 'new look'. The subject matter has been grouped along different lines to that followed in previous editions. The publishers hope that this arrangement will facilitate reference to subject matters. This may prove to be the case. I doubt the superiority of the present to the old arrangement; however, it may prove useful in other respects.

An alphabetical order is purported to be followed in presenting the contents but this is not consistently followed. Thus, 'Special Article' might have taken its place after 'Skin Diseases', and its subdivisions require arrangement alphabetically too. Similarly, the subdivisions under 'Alimentary Diseases' do not follow one another alphabetically giving the impression of haphazardness in their grouping which does not facilitate finding a particular point easily. The same applies to almost every other major subdivision in the book.

'Orthopaedic and Traumatic Surgery' might profitably have been grouped with and following 'General Surgery' under the heading 'Surgery', instead of being widely separated from each other.

On the medical side, some new articles appear: 'Changing Emphasis in Paediatrics'; 'Psychopharmacology' with special reference to pharmacological agents for influencing behaviour and aberrant mental and nervous states; a discussion of 'Carcinoma in Lung Scars'—a recent observation, and new ideas and advances in connexion with 'Nutrition and Vitamins'.

On the surgical side, attention is drawn to spectacular advances in cardiac, pulmonary, and gastro-intestinal surgery; the use of ileum in bladder and ureteric replacement in urology; the percutaneous vertebral angiography, and radiation dangers.

The book is packed with specialized information on all branches of medicine culled from British and American sources and, therefore, useful to specialists with here and there also something for the general practitioner. One would like to find more guidance in matters of treatment for the general practitioner, for he is more interested in this than in academical discussions.

G.C.A.v.d.W.

CORRESPONDENCE : BRIEWERUBRIEK

CAPE TOWN MEDICAL ART SOCIETY

To the Editor: The first exhibition of the Cape Town Medical Art Society will be opened on 31 March 1959 at 5.15 p.m. by Prof. B. Bromilow-Downing, at the S.A. Association of Arts (Small Gallery), Argus Building, Burg Street, Cape Town.

Members of the Cape Western Branch of the Medical Association residing in Cape Town are cordially invited to submit examples of their own paintings, drawings, sculpture, etc. for exhibition. Not more than 4 exhibits are to be handed to the Curator of the Gallery on Thursday 26 March. Typewritten labels should be attached to exhibits, giving the subject, material, and name of artist, in capitals.

All members of the Cape Western Branch of the Medical Association are invited to the opening.

C. W. Coplans
Hon. Secretary

906 Sam Newman House
28 Burg Street
Cape Town
6 March 1959

SOLID FOOD FOR BABIES

To the Editor: Although Dr. Levin[1] in his article on *Solid Food for Babies* discusses this 'new look' in infant nutrition from many angles, he has overlooked the *psychological* repercussions of feeding solids to very young babies. Numerous psychological theories postulate that the premature introduction of solids may lead to undesirable behaviour manifestations. A study by Childers and Hamil[2] clearly demonstrates how early feeding habits may affect the development of character and conduct.

A. A. Lazarus

41 Hillsborough Mansions
Cor. Pretoria and Klein Streets
Hillbrow, Johannesburg
2 March 1959

1. Levin, S. (1959): S. Afr. Med. J., 33, 149.
2. Childers, A. T. and Hamil, B. M. (1932): Amer. J. Orthopsychiat., 2, 134.

SOLID FOOD FOR BABIES

To the Editor: Those of your readers who still have doubts about the wisdom of feeding very young infants with solids, as described in your *Journal*,[1] may be interested in the following quotation from a recent address by C. A. Smith to the British Nutrition Society:[2]

'One school of thought in our country has now brought this practice (of feeding solids) into the second (and even the first) week after birth. Since we appear to export the more unusual manifestations of our culture to other nations (perhaps because they are surplus commodities) people in Britain may find themselves faced with attempts to introduce cereals, puréed vegetables, and even meats, into the neonatal diets over here. The difficulty (as with so many aspects of the adaptable newborn organism) is to prove to its advocates that such early feeding of solids may do harm and can do no particular good. Unfortunately, the mere fact of an infant's doing at a few days an act previously expected at 3-6 months is evidence—to some—that a useful advance has been accomplished.'

Some of your readers will recall a report in the daily Press[3] that an American child aged 2 years enjoys smoking 4 cigarettes daily. Do our infants perhaps require a similar quota?

E. Kahn

Baragwanath Hospital
Johannesburg
26 February 1959

1. Levin, S. (1959): S. Afr. Med. J., 33, 149.
2. Smith, C. A. (1958): Proc. Nutr. Soc., 17, 50.
3. *The Star*, Johannesburg, 25 February 1959.

MANAGEMENT IN CASES OF RAPE

To the Editor: May I congratulate you on a very reasonable reply to your questioner?[1]

There is a point, however, that requires clarification. After taking slides to identify spermatozoa and possible venereal infection from the victim, where does douching with suitable spermicidal agents end, and possible criminal abortion begin? Surely, if seen early enough, a dilatation and curettage with a flushing curette could only be a more thorough douche than a simple vaginal douche, and would be likely to remove any of the offending spermatozoa, at any rate up to uterine openings of the tubes.

The object of the operation would be, in effect, to remove foreign bodies forcibly inserted into the uterus against the patient's wish. If any spermatozoa had entered the tubes by then or, by an extraordinary coincidence, if an ovum were lying in the uterus ready to be fertilized, then, technically an abortion would have been effected. Surely, ethically, it is the intention that counts, and the chances against conception taking place within hours of the rape must be very great.

The emphasis is on immediacy of operation after the rape, and here the district surgeon and the general practitioner or gynaecologist could work hand in hand with the utmost speed. It would, however, be interesting to know what the attitude of the law and the various religious bodies would be to this procedure, as a routine.

L. E. D. F. Joubert
Medical Officer

Government Hospital
Mbabane, Swaziland
26 February 1959

1. Questions Answered (1959): S. Afr. Med. J., 33, 80.

DISTRICT SURGEONCIES

To the Editor: It is to be hoped that the impressive list of vacant district surgeoncies advertised in this week's *Journal* will make the Secretary for Health sit up and take notice.

As one who is about to vacate one of these listed posts for health reasons (if not for any other) I should like to point out to anyone interested enough to read these lines how the Department of Health is exploiting us.

For a salary ranging from £15 to £60 per month a district surgeon is on call 24 hours a day every day of the year. He may not leave his headquarters unless he has made suitable arrangements for a locum (for which he is not paid anything extra) at all times. He is not given an official telephone, neither is he paid an allowance towards rental for a telephone. He must pay for all official calls himself. He is paid certain allowances for certain duties but, taken over a year, this amounts to very little.

A district surgeon is paid 1s. per mile for travelling. Out of this he must pay for petrol and in these days it is mostly about 4s. per gallon. He must pay his garage bills and replace worn tyres. He must trade in his car every year and he loses an impressive sum on depreciation—all this at 1s. per mile.

It is eloquent of how hard up our profession is that the list of posts is not longer. May I warn the Secretary for Health that he is going to be in difficulties if urgent attention is not given to the remuneration of part-time district surgeons, especially also in view of the fact that a large number of doctors are forsaking general practice and taking up more secure and less strenuous posts in the full-time services.

'Never Again'

3 March 1959

[The following is an extract from the Minutes of a Meeting of Federal Council (M.A.S.A.) held in Durban in September 1957: 'At this stage Dr. Troskie stated that the District Surgeons' Group had raised a fund amongst themselves in gratitude for the work which Federal Council had done towards increasing district surgeons' emoluments. He handed in a cheque for £357 0s. 0d. together with a list of the donors (as a contribution to the Benevolent Fund of the Association.'—*Editor.*]

SOCIETY FOR CLINICAL AND EXPERIMENTAL HYPNOSIS

To the Editor: A meeting was convened by Dr. Maurice Herman in his capacity as South African representative of the International Society for Clinical and Experimental Hypnosis to investigate the formation of a South African branch of this Society. Interested individuals had previously been invited through the Correspondence Columns in the *South African Medical Journal* to communicate with Dr. Herman.

The meeting was attended by a representative section of the medical and dental professions. Dr. Herman gave a resumé of the events which led to the formation of the International Society and visualized the formation of a South African Society with similar high objectives, academic standards and ethical demands.

The matter was fully and enthusiastically discussed and it was agreed that initially the local society should assume an essentially academic form to stimulate the study, professional research, discussion, and publication of work pertinent to the scientific study of hypnosis and allied sciences. Only after the attainment of a satisfactory academic status, as demanded by the International Society, should affiliation to the Society be sought. Undergraduate instruction in hypnosis and the affiliation of recognized auxiliaries would be part of the future programme of the local society.

Two branches will be formed in South Africa initially; one, a Northern branch, with headquarters in Johannesburg to serve Transvaal and Natal, and the second, a Southern branch with headquarters in Cape Town, to serve the other provinces.

A steering committee was elected comprising the following office bearers: Dr. M. Herman, Chairman; Dr. M. Silbert, Secretary; and Drs. I. Mirvish, W. Steenkamp, A. Michael and S. Berkowicz additional members.

The International Society, the articles of which include Associate Membership, Full Membership, Fellowship and Honorary Membership, has recently granted the Chairman, Dr. M. Herman, the first Fellowship of the Society in South Africa.

It is hoped, in the near future, to conduct regular meetings addressed by leading persons in the field of hypnosis and allied sciences. Notice of these will be given in the medical press. All correspondence should be addressed to the Secretary.

M. Silbert
Hon. Secretary

7 Mimosa Arcade, Regent Road
Sea Point, Cape
5 March 1959

BACK ACHE WITHOUT NEUROLOGICAL SIGNS

To the Editor: I have found the item 'Questions Answered'[1] and your Editorial 'The Relief of Pain'[2] of considerable interest.

During the past 10 years I have been conducting an investigation into the possible place of manipulative procedures in general practice. The results of my investigation provide a much more satisfying answer to the question posed on the subject of 'Back Ache without Neurological Signs', than does that of your expert.

Firstly, may I point out that the first part of the answer is unnecessary since the question postulates 'healthy young men', who are not likely to be suffering from tuberculosis, ankylosing spondilitis, neoplasm or myelomatosis. In this type of case there would be deterioration in health which would remove them from the class of the 'acute' type.

Secondly, I do agree that the majority of the acute cases come into the mechanical group and that the increasing frequency of such lesions is to some considerable extent conditioned by persistent and recurrent faulty posture.

Finally, I entirely disagree with the treatment suggested in the majority of cases. Has your expert never considered the possibilities of manipulation in these cases?

It is at this point that I want to question certain remarks in your Editorial on 'The Relief of Pain'. Your attitude appears to be summed up in the phrase 'control of pain'. Surely the object to be aimed at is the *relief* of pain by the removal of its cause. This is particularly true in cases of pain of structural origin. Striving after the mere control of pain in these cases forms a parallel to the prescribing of rest in bed and salicylates for back ache without neurological signs.

Just before opening the *Journal* in which these items appear I happened to be checking on the subject of so-called 'rheumatic fibrositis' in a well-known surgical text-book.[3] I had in front of me two editions published 14 years apart. I found the matter in both identical (the page numbers differed). Torticollis is ascribed to 'exposure to draughts'. Lumbago, about which it is stated that 'the pain may be agonizing' is dealt with in 15 lines. For both conditions the suggested treatment is rest in bed and salicylates, 'though the patient may be allowed up to the toilet'. In lumbago a mustard plaster is advised. Is it any wonder that, in despair, many of our patients go to unorthodox practitioners? But, and this is important, can these unorthodox practitioners do something to *relieve* their pain by removal of its cause and, surprising as it may seem to the orthodox, in many cases the pain is relieved.

My records show that some 30% of the patients entering my rooms complain of pain which is relievable immediately, or almost immediately, by simple manipulative procedures which are well within the scope of the general practitioner.

Because of modern carelessness about posture, as your expert suggests, in driving motor cars, sitting at desks, and the increasing number of heavy objects which have to be picked up by untrained personnel, we are more exposed to trauma of the back. A pressure of hundreds of pounds on the lumbar vertebrae is not to be treated lightly.

'An imposing number of drugs is available . . . ' for 'the eradication of primary disease' of structural origin but it is certain that none of them can take the place of simple manipulation for the relief of acute structural abnormalities.

A more positive attitude to this aspect of medicine is long overdue and would result in a large number of structurally affected individuals returning to work in one or two days instead of one or two weeks and would remove the need for 'a more potent non-addictive centrally-acting analgesic', at least in the case of structural abnormalities.

Philip H. Dalgleish, M.B., Ch.B.

P.O. Box 79
Hill Crest, Natal
3 March 1959

1. Questions Answered (1959): S. Afr. Med. J., 33, 153.
2. Editorial (1959): *Ibid.*, 33, 155.
3. Romains, W. H. and Mitchiner, P. H. (1941): *The Science and Art of Surgery*, 7th ed. London: J. & A. Churchill.